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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

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Original Papers.

FURTHER EXPERIMENTAL WORK ON BACTERIALLY-PRODUCED NERVOUS TISSUE LESIONS.*

By

DAVID ORR, Edinburgh.

It has been demonstrated in a previous communication[1] that in lymphogenous infection of a peripheral nerve the lesion produced is interstitial in character; all the fixed tissues in contact with the infected lymph show a reaction which is of an acute nature at the site of infection focus, but becomes proportionately of a subacute and less active character in the portions of nerve more remote from the site of infection. The reaction is characterised by inflammatory phenomena, varying from a leucocytic and lymphocytic type to one in which plasma-cells are the predominant feature.

In the present series of experiments, in which bacteria have been introduced into the general circulation, it will be seen that the resultant lesion in the nervous structures is degenerative or destructive, and that it involves definite anatomical sites in the brain.

METHODS.

The entire brain was fixed in formol-saline 10 per cent., embedded *in toto* in paraffin, cut in serial section, and stained by the toluidin blue, Heidenhain's iron-hæmatoxylin, and Van Gieson's methods.

In the description of the nerve-cell lesions the term 'chromatolysis' will not be used, as the cells in the fornix, cornu Ammonis, and cerebral cortex have a small cell-body, and the chromophile material, normally, is of the finely powdered or amorphous variety. The term 'chromatolysis' or fragmentation of the chromophile material is therefore not applicable.

Coagulation-necrosis of the cell-body with homogeneous atrophy of the nucleus is, on the other hand, a very definite change, and easily recognisable by the intense affinity of the altered nerve-cells for stains, and their distortion and shrinkage. These appearances are indicative of active interference with cell nutrition, which is followed by necrosis and death.

* From the Laboratory of the College of Physicians, Edinburgh.

It is proposed therefore to adopt as a criterion of the lesion caused by the bacterial infection, the presence of the necrotic areas which were found in the fornix and cornu Ammonis.

The changes in the neuroglia have been, so far, observed in one experiment only. In order to preserve the complete anatomical continuity of the ganglionic laminæ in the brain, and as it was impossible to anticipate in which area of the cell laminæ a lesion might occur, the frontal pole was in each case chosen for special fixation of the neuroglial elements. Only in one instance was a necrotic area found to be included in this portion. The glial reaction will be studied more systematically therefore in another series of experiments.

DETAILS OF EXPERIMENTS.

RABBIT 1.

In the *fornix* of the *left* side there was a definite area of coagulation-necrosis affecting the nerve-cells of this structure in its *upper* portion. The necrosed area was situated at a point immediately inferior to that at which fornix becomes continous with frontal cortex, and was sharply defined from the adjoining healthy portion by its definite staining reaction. In the necrosed area the nerve-cells, with their nuclei, were very deeply stained, altered in outline, and shrunken, thus showing a combination of coagulation-necrosis of the cell-body with homogeneous atrophy of the nucleus. The nerve elements of the contiguous portion of the frontal cortex were, for a short distance, similarly affected.

On the *opposite side* of the brain (right), but at the inferior end of the fornix where it is continued into the uncus of the temporal lobe, there was an area of coagulation-necrosis similar in appearance to, and as sharply defined from the normal as, that in the superior portion of the opposite fornix. The necrotic change also involved the cells of the uncus at the tip of the temporal lobe, and could be followed over it along the pyriform lobe as far as the rhinal fissure, and for a very short distance beyond. The remaining portions of the fornix were normal save for a few minute areas of œdema scattered amongst the cells.

There was another area of necrosis in the *right fornix*, in addition to that above mentioned, situated in the zone occupied by the second quarter of the fornix nearest to the frontal cortex. The necrotic area showed the typical characteristics of coagulation-necrosis with homogeneous atrophy of the nucleus described above, and appeared as a definite blue-black band of necrosed cells which was slightly but appreciably narrowed in depth and divided by a sharply cut line of demarcation from the normal cells on either side, in which the clear nuclei formed a striking contrast with those in the morbid cells.

There were many minute areas of œdema throughout the fornix, and this morbid condition attained its maximum at the left superior and the right inferior extremity, causing a considerable degree of distortion of the tissue with fragmentation of the neurons which could be followed through many sections of the series. Just behind this level the nerve-cells in the fornix were normal except in that portion immediately in connection with the uncus. In this, and in the superficial layers of the pyriform lobe, there were many necrosed cells.

The *cornu Ammonis* showed morbid changes in both its outer or *granular* layer, and its inner or *pyramidal* layer.

There were many small discrete foci of *œdema* in both cell-layers, causing a definite destruction and almost total disappearance of the nerve-cells at the points affected. Many of the cell-bodies and nuclei of the *outer granular* layer stained much more deeply than normal and showed no detail of structure, an appearance indicative of coagulation-necrosis of the cell-body and homogeneous atrophy of the nucleus. All areas of this layer however were not equally affected.

In the inner layer of small pyriform cells there was the same necrotic change. The cell-body and the nuclei stood out in bold relief as small deeply stained structures which, in many instances, were distorted. The necrotic change did not affect this lamina uniformly but in a somewhat segmental manner, though not in so rigid a fashion as in the fornix.

At the *posterior extremity* of the fornix the nerve-cells on both sides were perfectly normal. The nucleus was clear and the scanty surrounding protoplasm showed no abnormal reaction. There were, however, numerous small areas of œdema scattered throughout its entire length.

In the posterior extremity of the *cornu Ammonis* the majority of the nerve-cells showed the hyperchromatism of homogeneous atrophy of the nucleus, and throughout the granular layer were many small areas of œdema.

RABBIT A.

The *fornix* was practically normal from its upper extremity in the superior cortex of the brain to its lower connection with the uncus of the temporal lobe; nor was there any cell change in the pyriform lobe. The cell-layer of the fornix was seen as an unbroken line of clear nuclei surrounded by a narrow zone of faintly stained cytoplasm, and its continuation below with the superficial cell-layer of the pyriform lobe as far as the rhinal fissure was normal also. The only morbid change present in the above structure was œdema, which showed as minute and irregularly scattered areas.

In the *cornu Ammonis* of the left side there were a few scattered degenerated cells in the inner or pyriform cell-layer at the upper pole, and likewise in the same layer at the inferior end. In both these regions of the opposite side the cell-layers were normal.

RABBIT B.

There was a high degree of general œdema of the brain which necessitated prolonged staining. The *fornix* had suffered severely, the œdema not only interfering with the staining reaction of the cells generally but destroying them in small patches. The areas in which destruction had actually occurred were more numerous and larger in the upper portion of the fornix, and had caused a definite circumscribed loss of nervous tissue especially in that region of the fornix which is continuous with the upper cerebral cortex. In the centre of these œdematous areas no definite tissue existed—merely debris—while the periphery consisted of compressed, distorted, faintly stained cells. No further points could be brought out here by iron-hæmatoxylin staining.

The *cornu Ammonis* was very œdematous also, and this had caused minute and larger areas of destruction in both cell-laminæ. The cells of both layers stained faintly by toluidin blue, thus resembling the reaction found in the fornix; but iron-hæmatoxylin staining brought out many scattered cells *of a small type** in the outer granular layers which stained heavily, especially in the superior half of this lamina. These necrotic granular cells were most numerous in the upper segment of the layer involving both margins for some distance, and the upper and lateral curve. The middle portion was affected to a lesser degree, while the inferior pole was practically normal. The necrotic zone occupied about one-third of the layer, its segmental character suggesting ischæmia as the mechanism of production. There were small discrete areas of œdema scattered throughout the necrotic zone which had led to a local loss of tissue. In the inner pyramidal layer there were a few scattered necrotic cells, and many small areas of œdema.

RABBIT C.

The brain tissue from this experiment was very œdematous throughout, rendering staining by aniline dyes a matter of considerable difficulty. In addition to this general

*Small diamond-shaped cells between the larger more vesicular-looking cells.

œdema there were many minute scattered areas of greater intensity which had caused minute circumscribed areas of destruction in the fornix, and in both granular and pyramidal layers of the cornu Ammonis.

In many of the œdematous areas either no nervous tissue could be defined, or the elements had been distorted and displaced. This applied to both the fornix and the cornu Ammonis. Where the elements were stainable by toluidin blue and had retained their form, both cytoplasm and nucleus were only faintly discernible and showed practically no detail of structure.

In the upper and mesial portion of the *cornu Ammonis* a few cells of the inner pyramidal layer were not quite so pale as the others, but their structure was very indefinite.

With the iron-hæmatoxylin stain many of the nerve-cell nuclei in the *fornix* stained a little more deeply and diffusely than normal, while the cytoplasm showed a similar change, giving to the cell a uniform grey black appearance in which the nucleolus alone stood out distinctly. In the outer or granular layer of the cornu Ammonis the nerve-cell nuclei, with very few exceptions, showed a marked affinity for iron-hæmatoxylin, and to such a degree that all detail of structure was masked by the diffuseness of the staining reaction. Owing to the œdematous condition of the tissue Van Gieson's stain proved of no value.

RABBIT D.

In this experiment the nerve-cell lesion was localised to definite sites in the fornix and cornu Ammonis, and was of a necrotic nature. The cells in the affected areas were distorted, shrunken, and showed a marked affinity for both toluidin blue and iron-hæmatoxylin, and formed a sharp contrast with the normal.

The *fornix* was affected on both sides, and at the same point, i.e., in the lateral curvature, midway between the upper and lower cortical regions. Above and below the necrotic area the fornix was normal, with the exception of a few cells at its inferior prolongation into the temporal lobe.

In sections taken at a more posterior level, towards the occipital pole, it was found that the areas of necrosis gradually disappeared. Each diminished in size though still preserving its definitely necrotic character; and it was observed that the lesion on the right side was more extensive than that on the left.

The *cornu Ammonis* on the left side showed little morbid change. The granular layer was quite healthy, but there were some scattered necrosed cells in the superior portion of the pyramidal layer. On the right side, a few of the cells in the granular layer were necrotic, but in the pyramidal layer there were three necrotic areas, two in the superior portion, and one at the inferior extremity, each separated by a small interval in which the nerve cells stained normally.

RABBIT I.

The *fornix* on the *right side* was quite normal, the entire structure showing as a continuous band of clear vesicular nuclei. The adjacent portions of the cortex, frontal on the upper surface and lower in the temporal lobe into which the fornix is continued, were normal also.

On the *left side* there was a small area of necrosis in the fornix situated at a point a little distance from its superior ending in the frontal cortex, and just opposite the superior pole of the cornu Ammonis.

This necrotic area was small, and occupied about one-fifth of the field under low power magnification (objective 16 mm. ocular no. 1) and was thrown into sharp relief by the normal clear vesicular cells on either side of it. All the remaining portion of the fornix was perfectly normal. The nerve-cells in the necrotic area were shrunken, deeply stained, and had lost all detail of structure.

In the *cornu Ammonis* there was a distinct difference in the staining reaction of the two sides. The pyriform layer on the right side was normal, but on the left side in the upper portion of this layer there were many necrotic nerve-cells situated at a point in the same

transverse plane as the necrotic area in the left fornix noted above. The remainder of the cells in this pyriform layer were normal.

The granular layer of both sides showed many cells of the small diamond-shaped type which stained intensely, and contrasted markedly with the larger and clearer nuclei of the vesicular-cell type normally found in this nerve-cell lamina.

RABBIT K.

There was a local lesion in the *fornix* of the *right side* involving its upper portion. It was sharply defined inferiorly from that portion of the fornix which sweeps towards the lateral aspect of the brain, but not quite so definitely on its internal aspect where the fornix cells join the mesial surface of the frontal cortex.

The cells in the necrotic area stained very deeply and both nucleus and cell-body were shrunken and distorted. In the outer portion of the necrotic area almost every cell was affected, forming a deeply stained and shrunken solid line; but more mesially the necrotic process had not affected all the cells of the fornix band, normal and necrosed elements lying side by side in a scattered discrete manner as far as the frontal cortex.

Immediately below this necrotic zone the fornix was quite normal for a definite segment of its course, but at a point mid-way between the upper and lower surface of the brain there was another necrotic area as sharply defined as the above. Here the degree of coagulation-necrosis of the nerve-cells was not quite so advanced as in the necrotic zone at a higher level; and the lesion did not involve the most inferior portion of the fornix where it approaches and is continued into the uncus of the temporal lobe. This latter zone was free from necrotic change.

In the fornix of the left side the cells were quite normal except for a few scattered necrosed nerve-cells at its inferior extremity and in the cortex of the uncus.

In the *cornu Ammonis* of the left side and in its inferior portion, the nerve-cells of the pyramidal layer were in the initial stages of coagulation-necrosis. Both nucleus and cytoplasm showed an increased affinity for the stain, and were diffusely coloured. On the right side there was a considerable number of scattered necrosed cells in the upper and more central portion of the pyramidal layer and in its middle segment.

If we contrast the two sides we find that in the right there are two definite clear-cut areas of 'mass' coagulation-necrosis which contrast sharply with the normal portions on either side of them, and with the normal fornix of the opposite side. The upper area of necrosis is of an earlier date, or of a more intense nature, than that of the lower, as shown by the deeper staining of its cells and their shrinkage. This shrinkage not only affects the individual cells but has narrowed the cell-band 'en masse' for a short distance.

SUMMARY OF EXPERIMENTAL RESULTS.

RABBIT 1.

Fornix—left: an area of coagulation-necrosis in the *upper* portion.

Fornix—right: an area of coagulation-necrosis in its *inferior portion*, continuous with necrosis of nerve-cells in the *uncus* of the *temporal lobe* extending to *rhinal fissure*.

A *third area* of coagulation-necrosis on the *right side* at a *point near the frontal cortex*. Minute areas of œdema causing cell destruction also present in both fornices.

Cornu Ammonis: in the outer granular layer, areas of œdema (minute): also coagulation-necrosis of nerve-cells in *both outer granular and inner pyriform layer*.

RABBIT A.

A slight degree of *œdema* of the *fornix*.

In the *cornu Ammonis* of the *left side* a few *necrotic cells* in the *pyriform* layer: on the *right side* both cell-layers of *cornu Ammonis* are normal.

RABBIT B.

A high *degree of œdema* with destruction of nervous tissue and a marked decrease in affinity for toluidin blue.

The *fornix* suffers severely from œdema, especially in its *upper portion*.

Cornu Ammonis very œdematous also, with nerve-cell destruction in *both* cell-laminæ.

With iron-hæmatoxylin, the nerve-cells of the *granular layer* in the *superior part* of the *cornu Ammonis* stain heavily. The *inferior portion* is *practically normal*. Many small areas of *œdema* amongst the cells of the *pyramidal layer*.

RABBIT C.

A *high degree of œdema*.

Small foci of destruction in both layers of the *cornu Ammonis*: staining reaction feeble with toluidin blue.

Cells of *granular layer* of *cornu Ammonis* show a marked affinity for iron-hæmatoxylin.

RABBIT D.

Fornix of each side shows a well defined *necrotic area* situated in the *lateral curve* of the fornix.

In the *cornu Ammonis* scattered necrosed cells in the inner *pyramidal* layer of the *left side*: *outer granular* layer healthy.

In the *right cornu Ammonis* the *granular layer* is *practically healthy*: the *inner pyramidal layer* shows three *areas of necrosis* separated from each other by a small interval of healthy tissue. The study of serial sections shows that the necrotic area in the fornix of the left side extends further posteriorly than that on the right.

RABBIT I.

Right fornix normal. In the *left fornix* a small *necrotic* area near its superior ending in the cortex.

Otherwise the brain shows no morbid change.

RABBIT K.

In the *upper portion* of the *right fornix* an area of *coagulation-necrosis* sharply defined from the normal below it. Immediately below this normal portion another *necrotic area*, as sharply defined as the above. The inferior portion of this *fornix* is *normal*.

In the *cornu Ammonis* of the *left side* the nerve-cells of the inferior portion of the pyramidal layer show the *early* stage *of coagulation-necrosis*.

The *cornu Ammonis* of the *right side* is *practically normal*. There are some scattered necrosed cells in the *central portion of the pyramidal layer*.

Coagulation-necrosis in rabbits I, D, J, K. In those there were necrotic areas sharply defined from the normal.

In *rabbits A, B, and C*, there was a *high degree of œdema.*

Definite *necrotic* areas therefore have been found in *four* of the seven brains examined ; in the other *three* there was a high degree of *œdema*, in spite of which, however, there is evidence of coagulation-necrosis in the heavy staining of the nerve-cells with the iron-hæmatoxylin method. It is reasonable to suppose that the presence of œdema has to a considerable extent interfered with the staining reaction in the last three cases, and masked the characteristic appearances of the necrotic change. Finally, it is worthy of note that there was no hæmorrhage at the site, or in the neighbourhood, of the necrotic foci in any of the experiments.

DISCUSSION.

In the cerebral cortex there are no gross lesions similar to the ischæmic softenings in the cornu Ammonis and fornix, but the nerve-cells exhibit varying degrees of coagulation-necrosis. A description of this morbid change has been already given, and the cortical cells show precisely the same morbid features and staining reactions so characteristic of this type of affection.

The coagulation-necrosis is not distributed in a uniform manner throughout the cortex. In the superior and lateral areas of the pallium the morbid change varies in degree from point to point, contiguous territories showing slight variations in intensity ; and all the cortical cell-laminæ are not involved in the necrotic change. For our present description we may divide the cortex from without inwards into : (1) molecular zone ; (2) external granular zone ; (3) pyramidal layer ; (4) internal granular zone ; (5) ganglionic layer ; (6) multiform layer. All these zones are not always distinct or present ; certain variations in cell lamination occur in different portions of the cortical field, but the layers as given above serve our present purpose.

The whole depth of the grey matter does not exhibit coagulation-necrosis of the nerve-cells. The morbid change includes all the outer layers as far inwards as the ganglionic lamina ; it is rare to find morbid nerve-cells in the deepest or multiform layer. In the outer layers the degenerated nerve-cells may be very numerous, or in fewer numbers, according as one passes from one point to another ; and granule and pyramidal cells are affected indiscriminately.

It is of importance to note that the above areas, viz., the cortex, fornix, and cornu Ammonis, are the seat of morbid changes, and that they are supplied by cortical vessels derived from the pia-arachnoid. We know that the central portion of the cornu Ammonis—the lamina involuta—is penetrated by folia of the pia-arachnoid accompanied by vessels. The affected areas therefore all possess one important point in common, viz., their blood-vessels are derived from the pial system, a fact which may explain the participation of the above-mentioned subcortical nuclei in the morbid process.

The morphological character of the lesions in the brain points very clearly to a disturbance of the circulation and therefore of nutrition. This, in the cerebral cortex, affects a wide area and finds its expression in the necrosis of nerve-cells in all strata, unaccompanied however by any local ischæmia indicative of complete vessel blockage. Evidence of a much more severe local nutritional disturbance is very obvious in the ganglionic and molecular layers of the cornu Ammonis and in the fornix.

A concise statement in reference to this subject will be found in an article on hæmorrhagic encephalitis by Bignami and Nazari[2], in which many important points are mentioned having a direct bearing upon this research. These authors have found that the lesions in the white and grey matter in hæmorrhagic encephalitis differ very materially, and their views regarding the cerebral circulation are based upon this. A sharp distinction is drawn between *miliary hæmorrhages*, which are characterized by their ring-like form round a necrotic focus and by their situation in the white matter, and the *hæmorrhagic infiltration* which follows thrombosis of the cerebral sinuses or meningeal veins. The hæmorrhage in this latter condition is extensive, affects the grey cortex, and decreases from without inwards. There are no necrotic foci surrounded by a ring of hæmorrhage such as occur in the white matter. According to the opinion of the authors both conditions are caused by a local disturbance of the circulation and not by an inflammatory process. In the one case the thrombosis of the cerebral veins induces hæmorrhage in the cortex from stasis ; in the other, blockage of a precapillary arteriole results in a circumscribed necrosis in the white matter followed by hæmorrhage into the immediately surrounding parts from the collateral vessels, the pathogenetic mechanism here being precisely the same as in infarction of other organs. If a similar lesion is not produced in the grey matter, through occlusion of the arterioles, it seems more than likely that there is some difference between the circulation in the cortex and that of the medullary substance. These observations incline the above-mentioned authors to the view that, although the precapillary arterioles in the white matter are physiologically terminal, those in the grey cortex are not. In the white matter occlusion of an arteriole is followed by infarction ; since the latter does not occur in the grey matter, evidently we must admit the possibility of an arterial anastomosis which is functionally adequate.

Many questions regarding the cerebral circulation are still only partially solved or remain obscure, but we are now in possession of certain facts which bear on the subject before us. All are agreed that the cortical arteries do not communicate with the basal arteries. Duret[3] regards the cortical arteries as terminal, but Cordiat and Féré[3] do not agree with this, although they grant that the anastomosis is not sufficient to permit of re-establishment of the circulation when obstructed by thrombus or embolus. Heubner[3] supports the view that numerous anastomoses occur amongst the pial arteries, but adds that

after their branches have penetrated the cortex anastomosis ceases. On this latter point he is supported by Duret and Beevor, who hold that the cortical arteries are terminal ; but, in spite of these definite statements, it would appear that the whole question is worthy of further study, since it has been shown that the myocardial arteries are not completely terminal in the anatomical sense, as has hitherto been held.

Of great importance in the field of pathogenesis is a knowledge of the relationship between the short cortical and the long subcortical arterioles. Both are derived from pial arteries. The short are purely cortical, while the long pass straight down into the white matter, where each supplies a very narrow territory owing to the small number of branches given off. Cortical arteries divide at once and very frequently, forming a fine capillary network which is richest in the deepest cortical lamina. Further, it would appear that the short cortical arterioles anastomose in the depth of the grey matter with the long or medullary branches. The important point to be specially noted, however, is that the cortical vascular network is far richer than that of the white matter.

Whatever our present knowledge may be, from the anatomical side, of the ultimate distribution and connections of the cerebral vascular system, we seem to be justified in assuming from morbid lesions that there is a difference in the two systems which subserve the grey and white matter respectively. The evidence of this is seen in the histological difference between the *cortical* and *subcortical* lesions. The former are diffuse and consist in necrosis of the nerve-cell units ; the latter—in the cornu Ammonis and the fornix—involve a circumscribed locality and are typical of infarction. As the arterioles which supply the cortex and the adjacent subcortical zone have a common origin—the pial arteries—the difference in type of the resultant lesions must depend upon the anatomical arrangements of fine capillary branches. The definite restriction of the subcortical lesion with its pathohistological elements can only be interpreted as ischæmic in origin, and secondary to blockage of a terminal artery. But the pathogenic mechanism of the diffuse coagulation-necrosis of the cortical nerve-cells presents a more complex problem and is very far from clear. From the character of this lesion we can say definitely that no infarction has occurred, and therefore the presumption might be advanced that cortical arterioles are not terminal. The individual nerve units alone have suffered, and in a manner which points to interference with their nutrition ; but the histological picture is far from what one associates with an ischæmia, and rather suggests a stasis due to blockage of veins, a deficiency of nutriment from narrowing of the lumen of arterioles and capillaries, or both combined, as is most probable. It is only some anatomical factor within the cortex itself which could explain this different type of lesion, and for the present one would be inclined to ascribe importance to the richness of the looped cortical vascular network, which may counterbalance the effects of interference with the vascular

supply. This seems to be the most reasonable view to take of a question which is still controversial, and stands in need of further investigation.

Von Braunmühl[4], in a paper which deals with the alterations in the brain in acute excitement, describes the changes in the nerve-cells and neuroglia especially ; as he remarks, these structures stand in such close relationship to the circulation, that they react at once to its disturbance. He has found that some regions may be hyperæmic, others anæmic—the capillaries in the hyperæmic areas being unusually wide and packed with corpuscles. There is a widespread stasis and prestasis, accompanied by fine capillary hæmorrhages. The nerve-cells are pale, and protoplasmic processes necrotic and granular.

The lesion, he considers, is due to a spastic ischæmia, followed by paralysis of the capillaries which accounts for their congestion. In the pyramidal layer of the cornu Ammonis coagulation-necrosis of the nerve-cells, and homogeneous atrophy of the nucleus, were present. In the ischæmic areas of the cortex, the same nerve-cell and nuclear changes were observed, accompanied by a secondary glial proliferation in the regions in which the dendrites and axons of the nerve cells had been destroyed. There was marked hyperæmia in the middle half of the cortex, but none above nor below this level.

The author considers that the above necrobiotic changes are induced by disturbances of the circulation, and adds that, in the absence of organic vessel changes, one should ascribe the necrosis to stasis of the circulation in some cases, in others to prestasis and diapedesis, and in a third group to ischæmias with more or less tissue-necrosis, all attributable to functional disturbance of the circulation. He adds that it is known that in acute excitement where there is a marked tendency to collapse, the central regulating mechanism of the vascular system is involved reflexly, causing irritation and preliminary narrowing of the vascular lumen, followed by, after some duration of time and further action of the irritant, a condition in which the vessels relax. The factors which bring this about are extraordinarily labile, and come into play, preferably, in connection with the vascular terminals. Their occurrence is facilitated the more frequent and more abrupt are the special demands on the circulation and its regulating mechanism. The author states very definitely, that in severe, long-continued, and fatal conditions of excitement of the most diverse origin, the whole vascular system, and especially the terminals, are injured ; that vasomotor oscillations occur which induce circulatory oscillations in the central nervous system, and therefore parenchymatous changes ; and that these occur perhaps with more regularity, the more acute and prolonged is the circulatory disturbance.

Von Braunmühl in another paper[5] ("Über Gehirnveränderungen bei puerperaler Eklampsie und ihre Enstehung durch Kreislaufstörungen"), returns to the argument. He emphasises the vascular factor, and especially the morphological changes in the vessels, as important in the causation of brain lesions, but points out that functional disturbances of the vascular system are

equally important. He draws attention to Neubürger's observation of ischæmic lesions in the cornu Ammonis and ascribes them to functional disturbance. He refers to the observation of Spielmeyer that this functional spasm plays an important part in the genesis of epilepsy, in which morphological changes are found in the cornu Ammonis and cerebellum. According to Neubürger and Metz, this functional factor comes into play in general paralysis, the poison acting upon the vessels of the central nervous system. A similar view is held by Wilders.

The central nervous system in Braunmühl's two cases of puerperal eclampsia affords ample evidence of ischæmic cell changes. In the first case the cortical nerve-cell bodies were necrotic and shrunken with homogeneous atrophy of the nucleus. In some cells this necrotic change was of slight degree, in others much more advanced. No definite change affected the neuroglia. The adventitia and endothelium of the smaller arteries and capillaries contained altered fat, especially near the necrotic areas. This naturally would be the degenerated products of myelin sheaths in the neighbourhood. Many of the vessel nuclei showed karyorrhexis. The changes in the cornu Ammonis were prominent. What the author terms 'ischæmic cell changes' were present in the pyramidal layer. The cell body was homogeneous, and the nucleus deeply stained. Œdema of the white matter was present. The cerebellum showed changes similar to those in the cerebrum, and no inflammatory phenomena were present.

In the second case, the features of an ischæmic lesion were even more prominent. The cortical nerve-cells were shrunken, angular, deeply stained, and their protoplasmic processes much more obvious than normally. Peri-cellular incrustations covered the cell-body, and throughout the tissue were small droplets of degenerated myelin. Similar changes affected the cells of the cornu Ammonis. Throughout the brain there were more or less distinct changes in the nerve-cells, especially in the nucleus dentatus.

The author, in his discussion on the nature of the pathological alteration in the nerve-cells, concludes that it falls into the category of 'ischæmic' or 'homogenising' cell disease, on account of the homogeneous and vacuolated appearance of the cytoplasm, and the intensely stained, badly defined, and structureless nucleus. Similar necrotic lesions in the cerebral cortex have been observed by Spatz and Neubürger, in whooping-cough, to occur in relationship with vessels. Some of those lesions were diffuse, others of a more focal nature.

It is noteworthy that the parenchymatous lesions attained a much higher degree in the cornu Ammonis than elsewhere. The nerve-cell processes stained with unusual intensity, and there were droplets of myelin along their course. In one case of acute eclampsia of short duration, the early signs of ischæmic cell change were found in Sommer's sector of the cornu Ammonis, while the remainder of the brain showed nothing unusual worthy of note. The cells of Purkinje in the cerebellum appeared normal, but those of the nucleus dentatus showed the 'homogeneous—ischæmic' change. There were no obvious

lesions of the vessels beyond wide dilatation of the capillaries with swelling of their endothelium. There were only a few necrotic areas.

Von Braunmühl concludes in his review that the nerve-cell changes are characteristic of ischæmia, and draws attention to the absence of any mesenchymal or glial proliferation. He admits however that this is an unusual appearance in necrosis, as it is common for the least injured glia to react. In this connection one would suggest that the poison which has produced coagulation-neurosis of the nerve-cells may have at the same time so injured the neuroglia in the neighbourhood that its potentiality for proliferation has been destroyed. With regard to the genesis of the lesions the author is strongly inclined to the opinion that such parenchymatous lesions are related to vascular disturbance, and, in the absence of thrombotic or other lesion, that this disturbance is of a functional nature.

In this connection the observations of Neubürger, in his work on angiospastic (non-embolic) infarction of the kidney, are extremely interesting. This, and gangrene of the extremities, he believes to be caused by functional vascular disturbance. Two other authors who support this view are Kuczinsky and Dosquet, who regard the presence of fibrin and few leucocytes in the lumen of the vessel as a sign of active intra-vitam vascular spasm.

Von Braunmühl inclines strongly to the view that the lesion is an ischæmic one, caused by a more or less active and wide-spread angiospasm of the cerebral vessels, followed by death of the nervous structures. He very rightly emphasises the importance of separating the diffuse from the focal lesions. In the cortex, the latter may affect a portion of the laminæ in such a way that diffuse changes alternate with focal, and he notes that where the glia has undergone proliferation there is evidence of neuronophage activity. In Sommer's sector of the cornu Ammonis, in which the pyramidal cells of this layer are rather loosely arranged, they are reduced to mere shadows, but high power magnification reveals that many of them still show signs of necrosis. Here the Hortega type of glial cells are hypertrophied, secondarily no doubt to destruction of the nerve-cells.

Von Braunmühl's view, briefly stated, is that the lesions in the brain are brought about by a primary spastic ischæmia, followed by paralysis and congestion of the capillaries. The nerve-cells of the pyramidal layer in the cornu Ammonis become necrosed, and the nucleus atrophies. There is a secondary proliferation and hypertrophy of the glia in the necrotic areas of the cortex in which the nerve-cells have lost their axons and protoplasmic processes. Here the necrotic lesions are of a diffuse type. In the pyramidal layer of the cornu Ammonis, however, where the necrotic change of the nerve-cells is of a high degree, the glial cells have been so injured by the noxious agent that their potentiality for proliferation has been destroyed. Under such circumstances only the least injured elements have retained their power of reaction. He adds that the homogeneous appearance of the nerve-cells is typical of an ischæmic lesion. This view is also held by Greenfield[6], according to whom the appear-

ances of coagulation-necrosis of a tissue are most definite, and the necrosed elements stain in a characteristic manner on account of their increased affinity for the dye. Each unit becomes transformed into a homogeneous mass of an increased consistence, and this occurs especially in regions where lymph is readily absorbed. In the initial stages of necrosis the cells are swollen and intensely stained; later, the colour fades. All the above appearances can in Greenfield's opinion be brought about by toxins.

There are other interesting points to be noted in regard to those lesions. Greenfield[6] has observed that in blockage of some cerebral arteries by embolism or thrombosis the collateral hyperæmia is rarely marked, and that hæmorrhage is rare. Further, the area of cortical softening is much smaller than that supplied by the obstructed artery, if the vessels are healthy. Even in total obstruction of one middle cerebral artery the softening may be limited to the parts supplied by its most central branches, the convolutions adjacent to the affected area obtaining sufficient collateral supply from other arteries.

In regard to the part played by the sympathetic nervous system in inflammation, he points out how dilatation of the vessels occurs even when all connection with the cerebospinal vasomotor centres has been severed. He infers from this that the injurious influence of the irritant must be exerted therefore either directly on the vessel wall, or by the intervention of a local vasomotor apparatus.

Müller[7] has demonstrated the presence of such a vasomotor apparatus in connection with the vessels of the pia-arachnoid, and those of the choroid plexus; and it would appear from his work that the nerve-supply of the vessels in the above-mentioned areas is very rich. In this connection it is therefore of interest to note that the vascular supply of the fornix and cornu Ammonis is carried inwards by a fold of the pia-arachnoid, the lamina involuta, which gives off vessels to both the above nerve-cell laminæ; and that it is precisely in those two situations that the well-defined ischæmic lesions occurred in the experiments on which this communication is based.

SUMMARY.

In conclusion one would briefly recapitulate the various factors which contribute to the formation of the lesions, and which are fully dealt with in the discussion.

These are the production of definite ischæmic lesions, or acute œdema, both of which lead to localised destruction of the nerve-cell layers and adjoining tissue. In simple coagulation-necrosis without œdema the lesion is necessarily less extensive and more restricted to the cell laminæ.

The localisation of the lesions ascribed to the fornix and cornu Ammonis may justify one in arguing that the vessels in those situations are terminal. In the cortex, where the lesions are less prominent and more diffuse in character, one may conclude that the absence of definite ischæmic lesions is due to the richness of the looped system of capillary supply.

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[4] Braunmühl, A. V., "Über Gehirnbefunde bei schweren Erregungszuständen," *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, cxvii, 163.

[5] Braunmühl, A. v., "Über Gehirnveränderungen bei Eklampsie und ihre Entstehung durch Kreislaufstörungen (zugleich ein Beitrag über weitere akute Kreislaufstörungen am Zentralnervensystem)," *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, cxvii, 698.

(These two papers give the references to the articles by Neubürger, Spielmeyer, Wilder *et al.*, mentioned in the text.)

[6] Greenfield, W. S., *Chapters in Pathology*, Edinburgh, 1905.

[7] Müller, L., *Die Lebensnerven*, Berlin, 1924.

EXPLANATION OF FIGURES.

Fig. 1.—Photograph of rabbit's brain to show: (*a*) Cornu Ammonis consisting of two layers, an outer of granular cells and an inner of pyriform cells; (*b*) fornix connecting the upper brain cortex with the lower where it is continuous with the cortex of the temporal lobe; note the rhinal fissure which marks the lateral extremity of the pyriform lobe.

Fig. 2.—Higher power view of cornu Ammonis, to show the lamina involuta surrounding it, containing a prolongation of the pia-arachnoid and vessels.

Fig. 3.—Shows the lamina involuta at the lower extremity of the cornu Ammonis.

Fig. 4.—(*a*) The fornix, in which there is a small area of coagulation-necrosis of the nerve-cells; (*b*) the granular layer of the cornu Ammonis.

Fig. 5.—A small area of coagulation-necrosis of the nerve-cells in the lateral portion of the fornix.

Fig. 6.—A higher power view of fig. 5. Shows the deep black staining of the nerve-cells with iron-hæmatoxylin: the normal cells stain grey.

Fig. 7.—Note the area of coagulation-necrosis in the fornix at the right and left sides of the photograph.

Fig. 8.—Coagulation-necrosis of nerve-cells of fornix at its lower extremity where it joins the uncus of the temporal lobe. "X" is an area of œdema.

Fig. 9.—Medium power photograph of fornix showing coagulation-necrosis of nerve-cells; the deeply stained and structureless necrosed cells contrast markedly with the normal in which the nucleus is clearly visible. Necrosed area at left side of photograph.

Fig. 10.—Shows the same appearances as in fig. 9. The nuclei of the norma cells are clear, whereas the cells in the neurosed portion stain deeply. The sharp demarcation of the necrotic zone is well demonstrated. Note the vessels in the lamina involuta, lying between the fornix and cornu Ammonis.

Figs. 11 and 12.—Magnified views of fig. 10, to show the striking contrast between the clear normal cells, with their pale vesicular nuclei, and the deeply stained shrunken necrosed neurones. In Fig. 11 the lamina involuta, with its vessels, is shown.

Fig. 13.—Note the coagulation-necrosis of the fornix cells at its lower end, where it joins the temporal lobe, and is continued to the uncus. "X" is an area of œdema.

Figs. 14, 15, 16.—Examples of glial reaction in the necrotic areas. Figs. 14 and 15 are taken from the upper, fig. 16 from the lower extremity of the fornix.

Plate I.

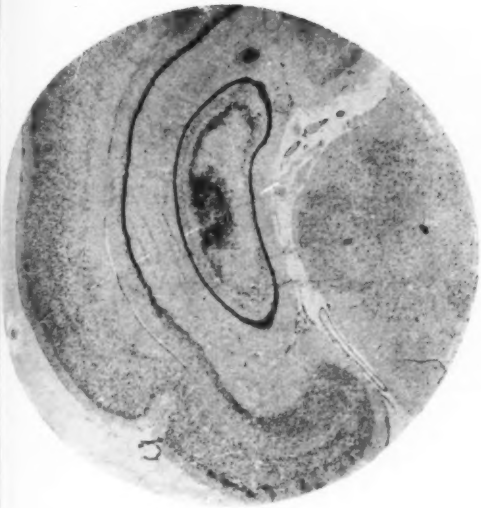

Fig. 1.

Fig. 2.

Fig. 3.

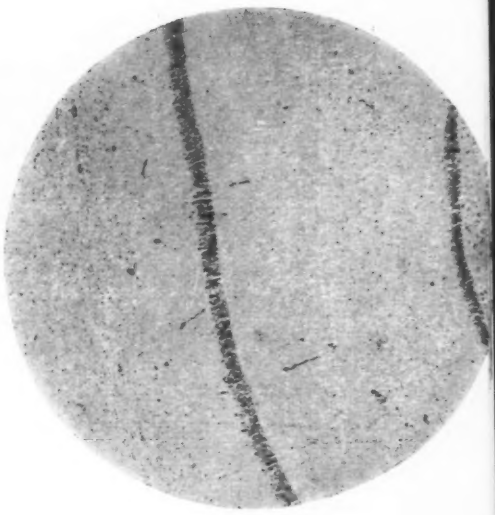

Fig. 4.

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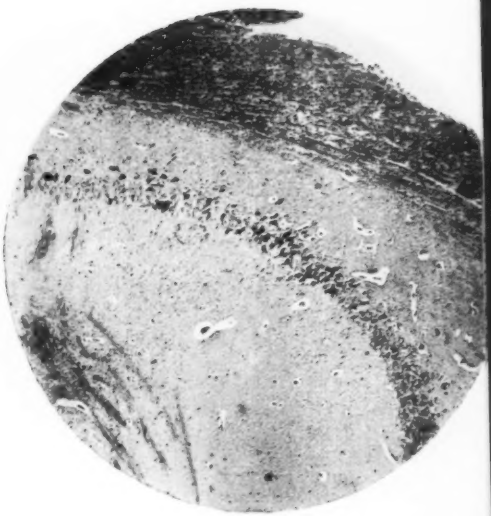
Plate II.

Fig. 5.



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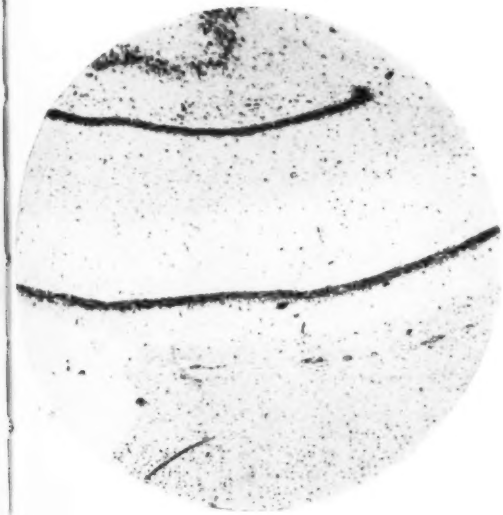

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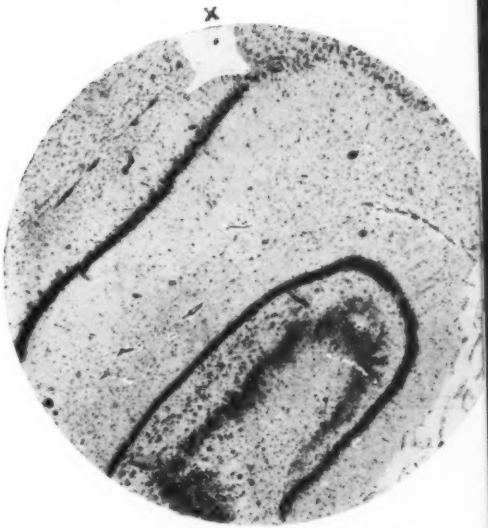

Fig. 8.

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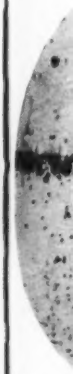

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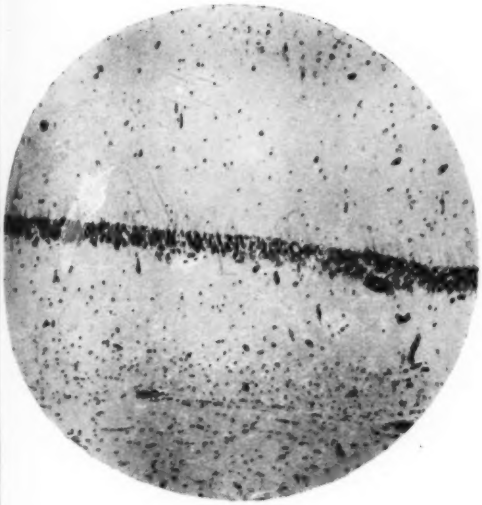

Fig. 9.

Fig. 10.

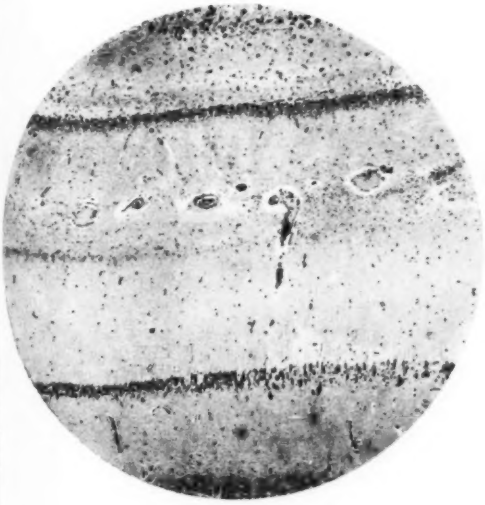

Fig. 11.

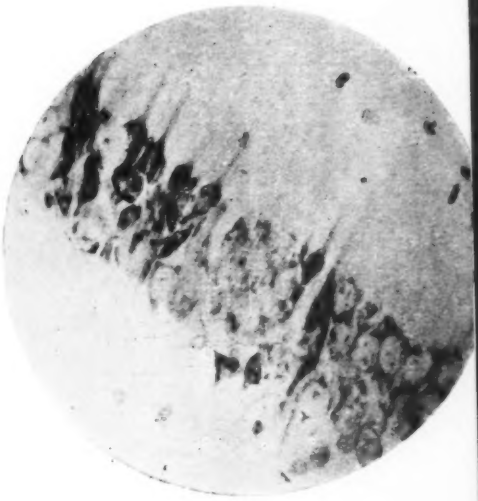

Fig. 12.

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Plate IV.

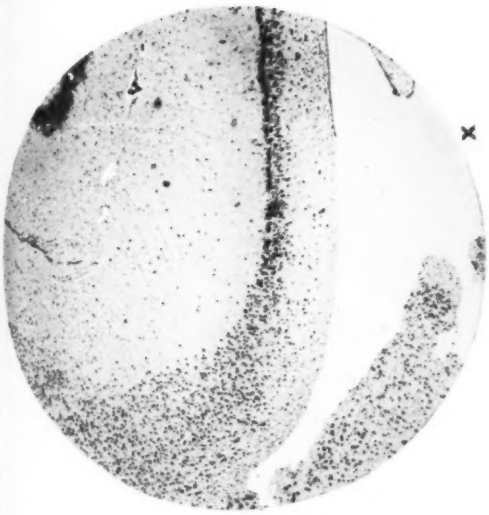
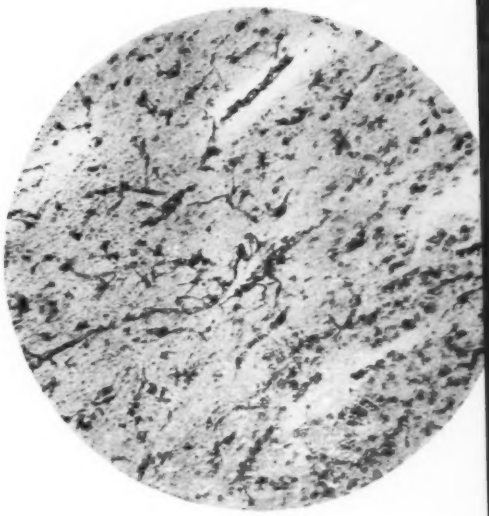


Fig. 13.



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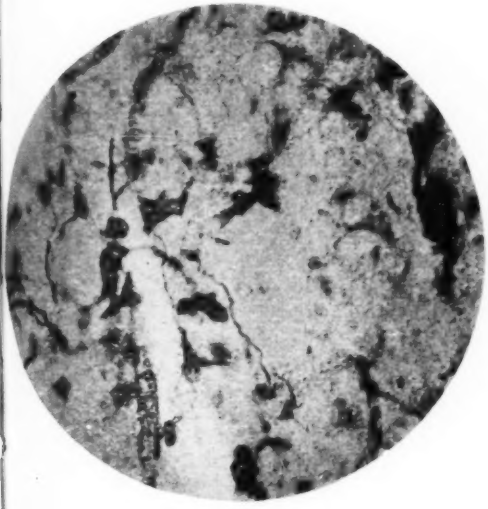

Fig. 15.

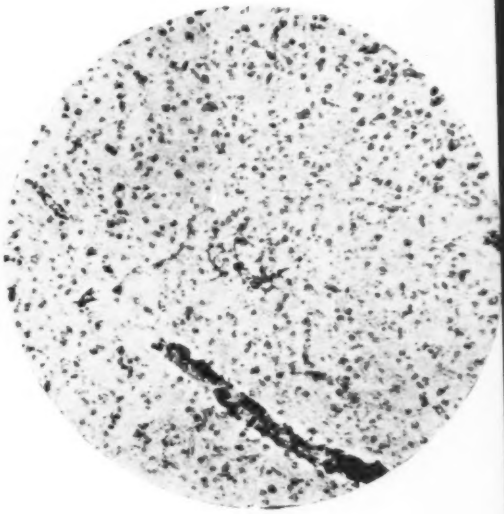

Fig. 16.

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TUMOURS INVOLVING THE CAUDA EQUINA: A REVIEW OF THEIR CLINICAL FEATURES AND DIFFERENTIAL DIAGNOSIS.*

By

I. M. ALLEN, London.

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I.—INTRODUCTION.

A general survey of the material available in the literature at once draws attention to the fact that it is only in the late and hopeless stages of cauda equina tumours that the classical clinical picture of a lesion of the cauda equina or even a collection of symptoms and signs similar to that produced by a traumatic lesion is encountered. An attempt has therefore been made to examine in detail the reports of cases available and to correlate the clinical and pathological findings with the diagnosis made at operation or at autopsy as well as with the localisation of the tumour itself. Such a review has naturally involved the consideration of the differential diagnosis of caudal tumours and this aspect of the subject has been dealt with in addition.

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The early recognition and localisation of tumours of the cauda equina are of paramount importance. In early cases successful removal and complete functional recovery are often possible, while in long-standing cases the chances of successful removal are much lessened and even when it is possible functional recovery is often infinitesimal.

The absolute frequency of tumours of the cauda equina is difficult to determine. It is possible to consider their frequency only in relation to that of spinal tumours as a whole. In a statistical study Steinke[1] found 30 cauda equina tumours in a series of 330 spinal tumours, an incidence of approximately 9 per cent. Elsberg[2] described 12 cases among 81 cases of spinal tumours, a frequency of 15 per cent. Parker[3] described 8 cases among 33 cases of spinal tumour, a frequency of 24 per cent. Such a high incidence was, however, rather counteracted by the occurrence among 85 cases of spinal tumour reported from the Mayo Clinic of only 3 cases with lesions of the cauda equina, only one of which proved to be a tumour. In general, it would appear from the statistics of the cases reported in the literature that approximately one in ten of spinal tumours affects the cauda equina.

The prognosis of cauda equina tumours is difficult to assess, more particularly as in such cases two separate factors must be considered, viz., (1) the chances of removal of the tumour, and (2) the prospects of functional recovery. An examination of the case records in the literature, however, makes it perfectly clear that the prognosis in relation to both factors is much improved if detailed investigation and surgical intervention are undertaken, in the one case as soon as a suspicious symptom appears and in the other as soon as unequivocal evidence of the presence of such a lesion is available. Tumours of the cauda equina are difficult to remove completely because of the number of nerve roots in that region, and because of the fact that the roots may pass through or surround the tumour, making careful dissection necessary for its removal. Of the 30 cases referred to by Steinke 13 or 43·3 per cent. were greatly improved and 14 or 46·6 per cent. died. Complete functional recovery occurred in 5 cases or 41·6 per cent. of Ott and Adson's[4,5] cases, a figure which probably represents the prospects of complete recovery in cases in which radical treatment is undertaken at a moderate period after the onset of symptoms. Lennalm[6] reported 15 cases in 3 of which a good result was obtained by operation, Cassirer[7] cured 2 cases out of 24 reported, Ehrenberg[8] had good results in 2 cases out of 3; Landelius[9] described 3 successful cases diagnosed before the onset of pareses or disturbances of sensation; while Karl Grosz[10] reported poor results in two cases of tumour of the cauda equina operated on late. Generally, with the exception of the more malignant tumours, good results have followed surgical intervention undertaken before definite pareses, pronounced sensory changes or serious disturbances of the sphincters appeared or in cases in which they were of short duration. The importance of the sphincters as a guide to the prospects of successful treatment will be dealt with at a later stage.

The type of tumour present would appear to have a considerable influence upon the prognosis, those which can be removed with as little injury as possible to important structures offering better prospects of success than those in which there has been extensive involvement of the nerve roots and of the conus medullaris.

II.—THE SYMPTOMS AND SIGNS OF LESIONS OF THE CAUDA EQUINA.

The classical descriptions of the clinical features of a cauda equina lesion are based upon a fairly extensive lesion, are applied in regard to some features to lower caudal lesions and in others to higher caudal lesions, and confuse the disturbances of the bladder and other functions produced by lesions of the sacral nerve roots and by those of the conus medullaris. Further, they presume the involvement of all the functions concerned and are, in consequence, scarcely applicable to the elucidation of cases of caudal tumours in which the functions may be affected in what appears to be a haphazard manner.

A more satisfactory working basis has been provided by Roussy and Lhermitte[11], who systematised the clinical features of traumatic lesions of the cauda equina in a study of the effects of gunshot wounds in that region. They differentiated five clinical syndromes :—

(1) a total syndrome, corresponding to lesions high up at the level of the second lumbar vertebra ;

(2) a lumbosacral syndrome, from lesions in the region of the lumbosacral articulation ;

(3) a sacral syndrome, due to lesions which are chiefly sacral ;

(4) a hemicaudal syndrome from lesions which attack one half of the cauda equina ; and

(5) a radicular syndrome, due to a lesion involving one or more of the nerve roots.

The total syndrome includes paralysis of the bladder and rectum, severe pain in the region of the sciatic nerve and in the sacral and perineal regions, loss of all reflexes in the lower limbs and anæsthesia of the whole of both lower limbs together with complete paralysis of all movements. It is based on a complete lesion of the cauda equina, involving all the lumbar and sacral roots below the exit of the second lumbar. The lumbosacral syndrome is the result of involvement of the fifth lumbar and all the sacral roots, and is described by Roussy and Lhermitte as the most common clinical type. It is characterised chiefly by paralysis of all the muscles supplied by the sciatic nerve on both sides, root pains in those areas, impairment of sensation in the distribution of the nerve roots involved, vesical and rectal troubles and absence of the ankle jerks. In a much less complete form it is a fairly common clinical picture in cases of caudal tumour. The sacral syndrome is characterised by root pains, vesical and rectal disturbances, saddle-shaped anæsthesia with sometimes impairment of sensation in a strip down the back of both thighs,

and the complete absence of motor troubles. It is described briefly as the ano-genital-vesical syndrome. The hemicaudal syndrome is characterised by the extreme preponderance of motor, sensory and reflex changes on one side, and is such as would be produced by a wound to one side of the middle line. Sphincter disturbances are usually transitory but occasionally they persist. The radicular syndrome varies in its details according to the root or roots affected. Monoplegia or paraplegia may appear after the injury and disappear; while only local limitation of movement, anæsthesia more or less in root areas, and various degrees of dissociation of sensation may remain. It is a clinical picture which resembles very closely that of caudal tumours in the early or intermediate stages when treatment can be carried out most effectively.

These syndromes, however, are based on lesions which are more or less acute and complete in themselves. In every case the initial damage is probably the maximum produced and the final clinical picture may be that of a definite, local circumscribed lesion. The syndromes are not applicable in toto to the study of lesions which arise insidiously and increase in their size and effect over a short or a long period. They take no account, moreover, of the peculiar features of caudal tumours in that they may damage some functions while others are unaffected. It would appear, however, that the hemicaudal and radicular syndromes might be applied with success to the study of caudal tumours in their earlier stages, while it is evident that the total and lumbo-sacral syndromes might be encountered fairly often in advanced and hopeless cases.

An attempt has been made to classify 69 cases of caudal tumour on the basis of the syndromes of Roussy and Lhermitte. While it was usually possible to fix the upper level of the lesion from the symptoms and physical signs and thus roughly classify the cases, it was nevertheless evident that, with the exception of the hemicaudal and radicular syndromes, the syndromes could not serve as a reliable working basis for the study of tumours of the cauda equina. Occasionally, in an advanced case the complete picture of the total syndrome was present but otherwise no syndrome could be regarded as complete. One or more important features of the syndrome were missing and no general conclusion could be reached as to how the clinical picture depended upon the localisation, extent and nature of the tumour. The only conclusion possible was that, when all the symptoms and signs were carefully considered, that produced by a lesion at the highest level probably gave an indication of the upper level of the tumour. Otherwise, the tumours appeared to affect certain nerve roots, leave others unaffected, interfere with some functions and leave others quite normal according to no obvious rule.

Wolfsohn and Morrissey[12] mention the possibility of applying the syndromes of Roussy and Lhermitte to the study of chronic lesions such as tumours and content themselves with stating that, though the syndromes are never so definite, they are sometimes helpful. With some reservations we can agree with this conclusion.

A study of the syndrome classification based upon the clinical evidence at the time of operation compared with that based on the pathological evidence reveals some interesting results. In the 'total' syndrome, particularly, the clinical picture was in nearly every case incomplete and in at least one-third of the cases some important clinical feature was missing. Clinically, the syndrome appeared to be 'total' in 64 per cent. of cases, 'lumbosacral' in 18 per cent., and 'sacral,' 'hemicaudal' and 'radicular' in 6 per cent. each. As was to be expected, in every case in which the clinical syndrome was of the 'total' type, even though incomplete, the pathological evidence confirmed the classification. In one half of the cases in which the syndrome appeared to be of the 'lumbosacral' type the lesion was of the 'total' type pathologically. An interesting conclusion is evident on comparing the frequency of the 'hemicaudal' and 'radicular' syndromes in the early stages of the illness with the frequency at the time of operation. In at least 91 per cent. of cases the clinical picture was of the 'hemicaudal' or 'radicular' type from a comparatively early stage, and in at least 61 per cent. of cases it was definitely of the 'radicular' type. This comparison at once suggests that, if the treatment of tumours of the cauda equina is to be satisfactory, the whole conception of the symptomatology must change so as to permit of complete investigation and treatment in the earliest stages. If that is to be so the usually accepted clinical picture of caudal tumours and even the syndromes of Roussy and Lhermitte must be put to one side as a basis for the study of tumours of the cauda equina.

The symptomatology of lesions of the cauda equina as applied to tumours in that region has been reviewed by Parker[3], Péron[13,14], Spiller [15] and others. The first emphasised the difficulty of studying tumours in this region on the basis of the recognised clinical syndromes. He found that tumours at widely separated levels may produce almost identical symptoms and physical signs and that, in some cases, the determination of the extent and position of the tumour seemed to be impossible. Every case appeared to differ. The type of tumour influenced the course, symptomatology and physical signs—the more malignant tumours producing a more rapid clinical course and more diffuse signs, while the slow-growing tumour produced a long course and clear-cut physical signs. An important source of difficulty was the fact that the sacral canal was wide and that a tumour might grow there for a long period without symptoms or physical signs. He reviewed the symptomatology and drew attention to certain clinical features which he had found to be of great value in diagnosis.

Spiller[15] reviewed the subject of tumours of the cauda equina up to 1908 and in particular stressed their differential diagnosis; while more recently Elsberg[2] has drawn attention to certain unusual features of tumours in that region. Apart from these the literature on tumours of the cauda equina has been confined to a review of individual cases and the discussion of their differ-

ential diagnosis. Exceptions have been a comprehensive paper by Ott and Adson[4] in which the symptomatology, diagnosis, localisation, pathology and the results of surgical treatment were discussed in detail, and another by Elsberg and Constable[16] in which the differential diagnosis of caudal tumours from neuritis of the caudal roots was considered.

III.—THE CLINICAL COURSE.

The duration of the clinical course of caudal tumours before coming under surgical treatment varies considerably with the nature of the tumour, the structures involved, the presence or absence of complications and the period of the clinical course at which the patient was first subjected to a thorough investigation. Of the cases referred to the average duration was about four years, the longest being 20 years and the shortest three months. It was particularly noticeable, however, that with the improvement in the knowledge of the initial symptoms and the use of special methods of investigation in those cases in which the initial symptoms were suggestive, the average duration of the clinical course before the patient came under effective treatment was much reduced. There always remain, however, numerous cases which have escaped investigation for several years because the condition has been regarded as sciatica, lumbago and other conditions. The duration of the clinical history varied considerably in the different series reported. The average duration of Spiller's cases was less than one year, that of Parker's three years, that of Elsberg's five and a half years, and that of Ott and Adson's a little over six years. The circumstances of practice and the material available vary so much that it is impossible to draw any general conclusion from these figures, excepting that a caudal tumour may produce a serious clinical picture in three months or twenty years from the onset according to the character of the tumour and the structures involved.

A study of case-records to determine the most important of the initial symptoms reveals the following information. *Pain* is an important initial symptom in 90 per cent., while in 10 per cent. it is not present as an initial symptom but appears at a later stage. In one case, that of Volhard[17], erroneously referred to as the only painless caudal tumour in the literature, the subsequent pain was rather of the nature of a feeling of pressure over the sacrum. In 70 per cent. *pain in the back* is the important initial symptom. It appears alone in 23 per cent., is associated with pain down one sciatic nerve in 10 per cent., and with pain down both sciatic nerves in 10 per cent. In 18 per cent. the pain in the legs develops at a later stage, so that initial pain in the back is associated with pain radiating into one or both legs, and chiefly into the area of supply of the sciatic nerve, in 36 per cent. *Pain in one or both legs*, either alone or associated with other symptoms, is the initial symptom in 61 per cent. It appears alone in one leg as 'sciatica' in 9·5 per cent., and alone in both legs as 'double sciatica' in 8 per cent. Occasionally, the pain in the leg or legs is confined to a portion of a segmental root area. In 1·5 per cent.

it appears in the foot alone ; in 3 per cent. it appears first in the hip in the region of the great trochanter ; and in 3 per cent. it appears in one gluteal region. In 3 per cent. it appears first in the back and one groin. *Pain in the lower part of the anterior abdominal wall* together with other symptoms is an initial symptom in 6 per cent. of cases, weakness of the legs appears first in 6 per cent. of cases, and interference with the functions of the bladder and bowel in 4·5 per cent. Unusual initial symptoms are pain in the rectum in 1·5 per cent., cramps in the legs in 1·5 per cent., and pain in the distribution of one anterior crural nerve in 3 per cent. Lower abdominal pain and interference with the functions of the bladder are initial symptoms in cases in which the tumour is at a high level and in which there is obviously some involvement of the conus medullaris or spinal cord. The importance of pain in the symptomatology and the relation of its situation to involvement of individual roots will be discussed at a later stage.

The clinical course of the condition varies considerably. Wolfsohn and Morrissey[12] describe the course as a rapid acute phase passing into a chronic or intermittent phase as regards the symptoms. The intermittency of symptoms in the early stages of caudal tumours, mentioned also by Beriel[13] and illustrated by many cases in the literature, is often a serious source of error in diagnosis. In about 25 per cent. of the cases symptoms appear for a short time and then disappear. This is particularly so when pain is the initial symptom. Sciatica appears on one side, is relieved and later reappears either on the same or on the other side. It is apparent, therefore, that the disappearance of the initial symptom and particularly of pain does not render a complete investigation of the patient unnecessary.

Péron[14] describes the characteristic clinical picture in three phases. The 'période de début' he ascribes to the early accident of irritation of nerve roots and the consequent production of root pains. At some time in their clinical course over 90 per cent. of cases show this clinical feature. The 'période de début' is characterised chiefly by root pains. Péron states that bladder symptoms often appear during this phase, but in doing so he is probably confusing the initial symptoms of caudal and of conus tumours. An onset with intestinal and rectal troubles is comparatively rare. The 'période de début' is the phase of diagnostic errors but none the less the phase during which the presence of a caudal tumour must be suspected and a complete investigation made if treatment is to be successful. Péron names his second phase the 'période d'état,' that during which definite symptoms appear and frank physical signs are present. These changes are one or all of : motor abnormalities including local or general flaccid paralysis and atrophy with variation in the corresponding reflexes, subjective and objective sensory changes, abnormalities of the pelvic viscera and of the sphincters, and trophic changes. It is the phase during which, on a complete physical examination, the diagnosis may be reasonably certain. The final phase described by Péron is that in which

the primary condition is so obscured by visceral and sphincteric disturbances that the patient's condition is more or less hopeless.

In general Péron's classification may be accepted as the basis of a working classification. The period of onset might be regarded as characterised by the symptoms described as initial symptoms. It is the period at which errors in diagnosis are certain to occur if the investigation stops at the stage of a neurological examination, the period at which special methods of investigation are necessary to make a complete diagnosis. The ease with which the initial symptoms are ascribed to conditions other than caudal tumours is largely responsible for the investigation being curtailed, but the success which has followed a complete investigation even in the earliest stages in cases described by Bennett[19], Wolfsohn and Morrissey[12], and by Cushing and Ayer[20] shows how necessary it is to carry out such investigations when the symptoms suggest a caudal lesion. In place of the term 'période de début' used by Péron this phase might be termed the 'phase of special investigations.' During the second phase in which definite symptoms and physical signs are present special methods of investigation may be necessary but are not essential for a working diagnosis. During this phase such investigations are most often needed for confirmation of the diagnosis and the exact localisation of the lesion. In the final phase, that of complications, the condition of the patient is usually hopeless, but that it is not always so after satisfactory treatment of the complications is shown by a case reported by Cushing and Ayer.

IV.—THE SYMPTOMS OF CAUDAL TUMOURS.

The initial symptoms of caudal tumours and the importance of pain as a factor in the early stages of the clinical course have been dealt with above. The symptoms are now described in detail and their particular characteristics stressed. Until recently the only case on record of a tumour of the cauda equina which did not give rise to symptoms or physical signs was that reported by Spiller[21] of a lipoma of the filum terminale, but systematic examination of the cauda equina at autopsy has shown that such silent caudal tumours are not uncommon.

(1) *Pain.*—Pain is the initial symptom in over 90 per cent. of cases of caudal tumour. In the remaining 10 per cent. it appears at a comparatively early stage of the clinical course. Volhard's[17] case which has been mentioned as being the only painless case in the literature was that of a patient who had fairly early a sense of pressure in the region of the sacrum. The time of appearance, localisation and distribution of the pain would appear to depend upon a series of factors such as the nature of the tumour, the rate of growth, the position of the tumour and particularly its antero-posterior relations to the roots of the cauda equina. It is apparent, however, that the last-named factor has a much greater influence upon the presence or absence of the objective sensory changes than upon the subjective sensory symptoms. Further, pain does not appear to be such an important symptom in cases in which a large soft

tumour fills up the greater part of the spinal canal as in those in which a small hard tumour is attached to the dura or to one of the nerve roots.

The position of the pain depends largely upon the caudal roots affected. In 79 per cent. of cases it appears first in the back, either in the lumbar or sacral region, and in 23 per cent. it never at any time appears elsewhere. In 10 per cent. it is associated with pain of the root type down one leg, and down both legs in a further 8 per cent. Eventually pain in both legs is associated with pain in the back in 36 per cent. of cases. It is evident from an examination of the case-records that pain in the back is in no way associated with involvement of the vertebræ by the tumour. Such an accident was comparatively uncommon in the records examined, whereas backache was an exceedingly common symptom. It is possible that the backache so common as an initial symptom may be a further manifestation of a nerve-root pain, but it is of little value in localisation because of the diffuse character of its distribution. In two-thirds of the cases in which it is present it appears in the lumbar region, and in the remaining one-third it appears in the sacro-iliac joint or in the sacral region. In the cases with lumbar pain the lesion was found to vary between the level of the fifth lumbar vertebra and that of the twelfth dorsal vertebra, the majority of them being towards the upper level. In the cases with lower back or sacral pain the lesion was in the lumbar region in one half and in the sacral region in one half. Spiller[15] states that the position of the pain in the back may sometimes be of value in localisation. From this review it is evident that the level of the pain in the back cannot be of great value clinically in fixing the level of the tumour. Such a pain is probably a referred pain from involvement of one or more of the caudal roots, or a local pain from involvement of the spinal meninges. In the former case it cannot always be certain that a nerve-root corresponding to the upper level of the tumour is involved. It might be supposed that the highest level at which the pain appears might help to fix the level of the lesion, but the vagueness with which pains in the back are usually described by the patient renders this almost impossible.

It is quite different, however, when the lower roots are involved and the pain is referred to the lower limbs. Pain in one or both legs is an initial symptom in 61 per cent. of cases. It appears alone in one leg as 'sciatica' in 9·5 per cent., and alone in both legs in 8 per cent. More often, however, it is associated with pain in the back. The common sequence is for pain to appear in the back or one lower limb and then to radiate into the other situation. The pain in the leg is usually referred down the back of the thigh and into the front or back of the leg. In some cases, at a later stage, it appears in the corresponding position in the other lower limb. In a few cases the pain appears in only a part of a segmental area, in the foot or in the hip. At first the pain is slight and intermittent and later becomes constant. Frequently it disappears for some months or even for a year and then reappears in the same situation in the same or in the other lower limb. The distribution of the pain is usually con-

fined to definite segmental areas, and frequently from its distribution the upper level at which irritation of the posterior nerve roots occurs can be estimated. When the lower sacral roots are involved the pain is referred to one gluteal region, to the back of one hip or to the perineum ; when the upper two sacral roots are involved, down the back of the thigh and leg ; and when the fifth lumbar root is involved it is referred to the front of the leg below the knee. In other cases pain is referred to the inner side of the knee, probably from involvement of the third and fourth lumbar root, and to the groin from involvement of the second lumbar root. In a few cases it is referred to the lower part of the anterior abdominal wall and occasionally to the iliac region.

The character of the pain and the conditions under which it occurs deserve some consideration. Generally, it has the characteristics of a root pain, but there are several important features in which it differs from a root pain occurring in lesions affecting the spinal cord and from that produced by a lesion of a peripheral nerve. The intermittency and the mild character of the pain in the early stages have already been referred to. It may appear during the later months of pregnancy and disappear after labour. Subsequently, it becomes severe and constant but even after it has been present in this form for a long period it may disappear and reappear only after a long interval. In one case the period of intermission was as long as seven years. The pain in the back is usually a dull ache and increases a little on movement of the back. Under such conditions the patient is often treated for lumbago. In some cases it is severe, has a burning, crushing character, and is referred to one or both sciatic nerve areas. Frequently there is a constant dull ache in the back and affected limb with acute exacerbations which the patient finds almost unbearable. Percussion of the spine, jarring of the head, stepping too firmly on the floor and sometimes moving the arm may cause an acute exacerbation. Coughing, straining and swallowing may aggravate the pain. In these respects the pain does not differ from ordinary root pains.

There appears to be a difference of opinion about the influence of movement upon the pain ; but the examination of a number of cases in which pain is present in the early stages only in the recumbent position and is completely absent even when walking many miles suggests that movement is not an aggravating factor, at least in the early stages. In two consecutive cases described by Parker[3] the pain was always worse in the recumbent position while in a further case reported by the same writer walking always relieved it. Parker further pointed out that in the early stages it was common for the patient to sit in a chair to get relief in preference to lying in bed, and stressed the fact that the pain associated with caudal tumours was usually relieved by movement. Lind and Lundstein[22] reported a case in which severe pain on the inner side of the thigh always became worse when going to sleep. In Wallgren's[23] case the pain was always worse at night, while in one of Wolfsohn and Morrissey's[12] cases the pain appeared only at night, increased in severity towards the morning and disappeared during the day. Bennett[19] described in

one case pain which was severe at night, and was present in the daytime only when the patient yawned or stooped. A similar contrast in the character of the pain has also been mentioned by Ehrenberg[24], Volhard[17], Wallgren[23] Lennalm[6], and Schmoll[25]. Hammes[26] and Box[27] particularly stressed the fact that the recumbent position aggravated the pain, the patient described by the latter being unable to sit or to lie comfortably on account of pain in the lower part of the back. Viets'[28] patient could sleep only in a chair, while Schmoll's patient was obliged to sit in a chair and rest his head on the side of the bed in order to get sleep. In their review of caudal tumours, Ott and Adson[4] stressed this contrast in the severity of the pain as an important factor in the differentiation of the pain due to irritation of the roots of the cauda equina from that of sciatica. Occasionally a case has been reported in which the pain has been aggravated by movement but nevertheless the greater part of the evidence goes to show that the pain of caudal irritation due to tumours is aggravated in the recumbent position and relieved in the erect position and by movement.

An interesting explanation of this variation has been offered by Wallgren, who has suggested that the contrast is due to the varying degree of tension upon the roots of the cauda equina in the two positions. His explanation is that in the recumbent position the normal lumbar lordosis is decreased, the lumbosacral canal is lengthened, and the tension thus put on the caudal roots already subjected to irritation is sufficient to lead to severe exacerbations of pain. Conversely, the erect position increases the lumbar lordosis, lessens the length of the spinal canal and the tension on the caudal roots and thus relieves the pain. There is, however, the possibility that in some cases the lessening of cortical control with the inducement of sleep is responsible in some measure for the onset of the pain at night. The onset of sleep has been noted as an important factor in those cases in which the nerve-roots have been subject to irritation because of osteoarthritis, as they passed through the intervertebral canals. Variation in the lumbar curve has of course no influence in such cases. Such a contrast may sometimes be of value in differentiating pain due to irritation of caudal roots inside the spinal canal in the vertical part of their course from that due to irritation of the nerve trunks as they pass through the intervertebral foramina—in fact, between an intraspinal and a spinal condition. The pain of sciatica is increased by movement and lessened by rest and the recumbent position, an observation in direct contrast with that usual in caudal lesions. The contrast with the root pains due to lesions of the spinal cord is not always evident.

Even though the area of supply of a lumbar or sacral nerve-root or roots has become completely anæsthetic, pain may continue to be felt there—a condition of anæsthesia dolorosa. In such cases a number of different sensations may be felt in the whole or part of the segmental area. The explanation probably is that, while the roots are so destroyed that sensations from the

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periphery are conducted very poorly or not at all, the lesion still serves as a source of stimulation to the central fibres and the impulses so initiated are interpreted in different ways by the central mechanism.

(2) *Other spontaneous sensations.*—Pain is by far the commonest sensation in the area affected, while other forms of spontaneous sensation are comparatively uncommon. Occasionally, a feeling of numbness is felt in part of the segmental area in which the pain appears and sometimes at the same time as the pain. Sometimes a burning or a pricking sensation is felt in one leg or in the foot. Numbness or a burning pain is sometimes felt in the perineum. Numbness and tingling in one foot occurred in one case reported by Elsberg[2]. It is unusual, however, for the patient to be aware of any change in sensation. A cramping sensation sometimes occurs in one or both legs, and a feeling of fulness or of something in the rectum was present in one case.

(3) *Sphincter disturbances.*—Of the cases in which at operation or at autopsy a pure cauda equina lesion is found with no involvement of the conus or of the lumbosacral cord the sphincters are normal throughout in 34 per cent. In 50 per cent. of cases both bladder and bowel are affected at some time during the clinical course, the bladder alone being affected in 20 per cent. and the bowel alone in 2 per cent. Increased frequency of micturition is the initial symptom in 4·5 per cent.

A study of the sphincter disturbances in relation to the period of the clinical course reveals the following interesting information. In the early stages of the condition, in which is included the first half of the clinical course, retention of urine occurs in only 5 per cent., frequency of micturition in 4·5 per cent., while incontinence does not occur at all. During this stage, disturbance of the bowel in the form of constipation is present in less than 1 per cent.

During the second half of the clinical course sphincter disturbances occur much more frequently, frequency of micturition in 2 per cent., retention of urine in 41·5 per cent., and incontinence in 34 per cent. Constipation appears in 34 per cent., and lack of control of the bowel in 35 per cent. As for the bladder the usual sequence of events is frequency of micturition followed by or associated with retention of urine, and at a later stage by incontinence. With the bowel constipation usually precedes lack of control of the bowel movements. Nevertheless, as indicated above, disturbances of the sphincters are entirely absent in 34 per cent.

Reference to the anatomy and physiology of the nerves supplying the bladder shows that a pure caudal lesion can produce only two effects, viz., (1) interruption of the sacral autonomic path by involvement of the second and third sacral roots ; (2) interruption of the afferent stimuli from the mucous membrane of the urethra. Physiologically, the sequence of events from involvement of these parts would be in turn (1) frequency of micturition ; (2) retention of urine from paralysis of the detrusor with a slight degree of

automatic emptying of the bladder ; and finally (3) incontinence. A similar sequence of events might be expected in the bowel. From the small number of nerve roots to be involved and the habit of caudal tumours of involving some structures and missing others, it is apparent that the chances of the sphincters being implicated in the course of caudal tumours are no greater than that of involvement of any individual group of nerve fibres.

In one group of cases incontinence of urine and lack of control of the bladder appear early in the clinical course or comparatively suddenly after various symptoms have been present for a long period. In every one of these cases, either at operation or at autopsy it is found that the conus has been involved by the tumour ; so that it is reasonable to assume that, if these symptoms appear rapidly at any stage of the clinical course, it is probable that the conus has become involved. Such an accident means the difference in treatment and prognosis which depends upon dealing with a structure in which regeneration or functional recovery may be impossible and one in which they are possible. The rapid onset of such symptoms must, therefore, be regarded as a factor of grave importance in the prognosis of caudal tumours.

(4) *Motor disabilities.*—Weakness of the legs occurs as an initial symptom in 3 per cent. of cases and occasionally the clinical course is initiated by the rapid onset of paralysis of both legs. In about 30 per cent. of an average series of cases there is no motor disability whatever. In the remaining 60 per cent., it is practically always preceded by sensory symptoms and is frequently associated with disturbances of the sphincters. Usually the first complaint is of difficulty in walking or of weakness in one or both legs. Sometimes a history of local weakness is elicited, of weakness above or below the knee or of drop-foot. As a rule the difficulty in walking and the weakness of the legs become gradually worse. Occasionally the onset of motor symptoms is sudden and the legs become affected so rapidly that the patient is soon unable to stand or to walk. This is, however, somewhat unusual. In those cases in which the disability is confined to one group such as the muscles in the leg supplied by the sciatic nerve the weakness may not progress further.

Certain other motor symptoms are sometimes present. Spasm of the erector spinæ muscles and sometimes of the hamstrings has been described by Bennett[19]. Stiffness of the back is sometimes complained of by the patient. Cramps in the legs especially on going to sleep occur occasionally. Rarely some difficulty in walking is described but no associated weakness of the legs is found to account for it. Local wasting is rarely referred to by the patient, though on being questioned he may admit that one leg is smaller than the other or that both legs are getting thin.

(5) *Miscellaneous symptoms.* Loss of the power of erection and later of ejaculation usually appears late and apparently progresses pari passu with disturbances of the sphincters.

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V.—THE PHYSICAL SIGNS OF CAUDAL TUMOURS.

In cases examined during the period of onset the physical signs may be very scanty and special methods of examination including the investigation of the cerebrospinal fluid may be necessary in order to make or confirm a diagnosis of caudal tumour. In the average case which has reached the 'période d'état' the physical signs elicited may be grouped and described as follows :—(1) general abnormalities ; (2) changes in sensation ; (3) variations in the motor functions ; (4) variations in the reflexes ; (5) variations in the organic reflexes and associated functions ; (6) the results of X-ray examination ; (7) changes in the cerebrospinal fluid ; and (8) the results of special methods of investigation.

(1) *General Abnormalities.*—The most important of the general abnormalities are those associated with the vertebral column. The lumbosacral spine may be stiff and the erector spinæ muscles hard and firm. Flexion, extension and rotation of the spine may leave the lumbosacral portion quite immobile and produce an exacerbation of pain referred to an area in the lower limb. Pressure on the spine itself may elicit tenderness in the lumbar or sacral region and aggravate root pains already present in the lower limbs. Pressure on the top of the head may again produce local pain and exacerbation of root pains. Partial obliteration of the lumbar curve is present in some cases and may be due to a compensatory mechanism to relieve as far as possible the tension on the caudal roots. Ott and Adson[4] observed local tenderness of the spine in over one half of their cases. These signs are particularly important as they resemble very closely those elicited in local conditions of the lumbar muscles and of the lumbosacral spine.

(2) *Variations in Sensation.*—In about 23 per cent. of cases no objective sensory changes are found. Sometimes this may be so only in the early stages, but sometimes such changes are still absent when variations in the motor functions and the reflexes are fully developed. Such an apparent discrepancy between the motor and the sensory changes may often give a clue to the position of the tumour in relation to the caudal roots ; but, while occasionally in such cases the tumour is anteriorly placed, this is not always so. The remaining cases can be explained by the apparent habit of caudal tumours of involving some structures and leaving others unaffected.

In the great majority of cases the superficial sensations are affected and not the deep sensations. Of the cases reported in the literature tactile, thermal and pain sensibility was affected without involvement of the deep pressure and vibration sensation and the sense of position in 85 per cent. In a few cases tactile sensibility alone is affected, and in a few others tactile and pain sensibility alone. In an occasional case pain sensibility alone or pain and thermal sensibility together are affected ; but as a rule such findings are due to the fact that pain and thermal sensory abnormalities are more readily elicited and defined than are those of tactile sensibility. In only a rare case are pain and thermal sensation affected to the exclusion of variations in tactile sensation,

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a finding which refutes the claim made by some writers that a dissociation of sensation of the syringomyelic type occurs in pure caudal lesions. Occasionally it is found that the areas of tactile, pain and thermal loss differ somewhat but not to any great extent, while the different types of sensation may be affected to different degrees. In the early stages the sensory changes may be slight and ill-defined; and as the condition develops all changes from slight loss to complete impairment may be encountered. As far as can be judged, the order in which superficial sensations are affected is probably tactile sensation first and thermal and pain sensation afterwards, though it is probable that in most cases all are more or less affected at the same time.

Variations in the deep sensations—deep pressure sense, vibration sense and the sense of position—apparently do not occur in the absence of variations in the superficial sensations, and in fact may be presumed to follow them. Once they are affected the variations in the deep sensations apparently proceed parri passu with those in the superficial sensations. In general, loss of vibration sense and of the sense of position appears to precede a loss of deep pressure sense, a sequence which has been suggested by Parker[3] as an important point in the differential diagnosis of caudal tumours and sacral tabes dorsalis. In many cases, objective changes in the sensations proceed pari passu with changes in the motor functions and in the sphincters, but to Parker's claim that this is usually the case we are unable to subscribe, as the position of the tumour in relation to the caudal roots and the character of the tumour itself lead to a pronounced discrepancy between the two groups of findings in more than one-third of the cases.

The sensory changes are usually found in the segmental area in which the pain occurs and in those adjacent to it. For example, it is common to find sensory changes in the first and second sacral segmental areas when pain is present down the back of the thigh and leg. Minor variations may be present in the segmental area immediately above and that immediately below that which is most affected, but the often quoted statement that the characteristic sensory changes occur chiefly in the third, fourth and fifth sacral areas (saddle anæsthesia) cannot be sustained. It is true that with pure sacral tumours the saddle area is often the first and only part affected and that when sensory changes are found in both lower limbs they may occur symmetrically in this area, but to state that it is the general rule is inaccurate. Probably such a generalisation had its origin in the artificial symptomatology deduced from the anatomy and physiology of the caudal roots, or in the practice of basing the recognised clinical features of caudal tumours entirely upon those of traumatic lesions of the cauda equina. Variations in sensation in the saddle area, even when they appear after sensory changes elsewhere, are found in less than one-third of the cases of caudal tumour. Asymmetrical changes in sensation are found in 60 per cent. of the cases. Symmetrical changes are more common when the abnormalities are found in either the upper or the lower sacral areas, that is in the feet or in the saddle area. In those cases with asymmetrical

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changes, variations in one or more of the lower lumbar segmental areas are usually present and at a later stage the saddle area may become involved on both sides. Though theoretically tumours reaching a higher level than the sacral roots might be expected to produce extensive and possibly symmetrical changes, in practice this is not found to be the case. Involvement of the saddle area and symmetry would appear to be associated with low-placed tumours, while asymmetry with or without involvement of the saddle area would appear to be characteristic of high-placed tumours.

Occasionally the upper level of sensory loss is found to have some direct relation to the upper level of the tumour, but in the majority of cases this factor is an inaccurate guide.

In rare cases other sensory abnormalities may be present Sometimes the margins of the anæsthetic areas are hyperæsthetic, as in one case reported by Parker[3]. Local tenderness of the lumbar spine and pain referred to one or both legs on palpation have already been mentioned Pressure over the great sciatic nerve below the gluteal fold and even pressure on the external popliteal nerve behind the head of the fibula may elicit marked tenderness. Rarely paroxysms of pain are initiated by movements of the thighs or by pressure. These, however, have been reported in only one case, that described by Box[27]. The muscles of the lower limbs and especially of the calves are rarely tender to pressure ; and very rarely flexion and extension of the knee produce pain in the distribution of the sciatic nerve.

(3) *Variations in the motor functions.*—Variations in the motor functions appear long after the subjective sensory changes. The time relation to objective sensory changes varies considerably. Sometimes they precede and sometimes they follow them. Frequently they are entirely absent. When present the segmental areas involved do not necessarily coincide with those in which sensation is affected. Sometimes they are at a higher level and sometimes at a lower level. The changes may appear on one side or on both sides. Occasionally they are symmetrical but more often they are asymmetrical. Most often there is weakness of an individual movement of one joint but no definite changes can be detected in one muscle or group of muscles. When more advanced there is paresis with atrophy and flaccidity in one group of muscles while those corresponding to the segments above and below are often weak. The group of muscles affected depends entirely upon the motor nerve roots involved. The group of muscles most often affected is the peroneal and less often the anterior tibial, resulting in each case in some degree of drop-foot and some weakness of dorsiflexion of the foot. Less commonly the posterior tibial muscles and the hamstrings are affected. Occasionally only the gluteal muscles on one or both sides are involved, leading to wasting over the sacrum and flattening of the gluteal fold and thus adding to the risks of a diagnostic error. Occasionally there is general weakness of one or both legs without any definite evidence of local paralysis. The quadriceps group is less commonly affected than are the other groups. When the tumour is placed low down in the

sacral canal the motor functions may escape entirely or the gluteal muscles alone may be affected. When several motor roots are involved there may be extensive flaccidity of one or both legs below the knee or of one or both lower limbs below the hip joint. Such extensive involvement of muscles is, however, exceedingly uncommon with caudal tumours. It is much more common for one or more segmental groups to be affected and for the others to escape.

Unusual features sometimes appear. Bennett[19] has reported a series of cases with spasm and contracture of the hamstring muscles associated with spasm of the erector spinæ muscles and rigidity of the spine. Fibrillary contractions are not uncommon in the affected muscles and a more or less definite reaction of degeneration may be present.

The gait varies considerably when the muscles are affected. Usually the patient walks weakly. However, when special groups are affected, certain characteristic types of gait are present. When the peroneal and anterior tibial groups are affected the gait may be of the steppage type. Sometimes it may appear to be definitely ataxic.

The importance of bilateral involvement of the motor functions in a case suspected to be one of caudal tumour cannot be underestimated, since in many cases of so called sciatica it may be the only clinical feature directly pointing to a lesion of the caudal roots. As an indication of the level of the lesion motor involvement has the same indefinite value as the subjective and objective sensory changes. The level of motor involvement may, however, indicate that the cauda equina has been affected at a higher level than is indicated by the changes in sensation.

(4) *Variations in the reflexes.*—The condition of individual reflexes in caudal tumours depends entirely upon whether the particular nerve fibres supplying the reflex are affected or not. In approximately 10 per cent. of cases, including many in an advanced stage, the reflexes are entirely unaffected. Péron states that the reflexes in the sacral area are affected early, the ankle jerks and the hamstring reflexes being usually the first to disappear; and further that the knee jerks are usually present but are diminished or lost if the lesion spreads upwards. Similarly, current descriptions of the symptomatology of caudal tumours usually include the statement that the ankle-jerks are lost, the plantar reflexes give no response, while the knee-jerks and cremasteric reflexes are usually present. Such general statements are not supported by clinical experience. They take no account of the presence of lesions at a high level and of the fact that a tumour at a high level may involve one or more caudal roots and leave the others unaffected.

The reflexes are affected unilaterally or bilaterally according to the lateral position of the tumour. Frequently the changes are unilateral at first and become bilateral as the lesion extends. In an intermediate stage the changes are often asymmetrical but as motor and sensory changes in the lower limbs progress they are apt to be bilateral and symmetrical.

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The first reflex to be affected depends entirely upon the caudal roots first involved. In the majority of cases an abnormality of the ankle jerk is noted first. One may be diminished or absent and the other remain normal; one absent and the other diminished, or both absent. In a minority of cases the knee-jerk may be affected first. One may be diminished or absent or both may be absent before the other reflexes are involved. Rarely one cremasteric reflex may be absent before the other reflexes are affected. In a case reported by Wallgren[23] both knee-jerks were absent and both ankle-jerks still present. In one of Bennett's[8,19] cases one knee-jerk was absent and the other reflexes unaffected in the early stages, while at a later stage both the knee- and ankle-jerks on that side were absent.

When one limb is affected the ankle- and the knee-jerks on that side may be absent together and the reflexes in the other limb unaffected. Occasionally in such a case the knee- and ankle-jerks in the opposite limb are exaggerated. Sometimes the ankle-jerk on one side and both knee-jerks are absent. In a case reported by Elsberg[2] both ankle-jerks were absent, both cremasteric reflexes absent, one knee-jerk diminished and the other exaggerated. At a later stage both ankle-jerks may be absent with absence of one or both knee-jerks, and finally both ankle- and both knee-jerks may be absent. In Ott and Adson's series of 12 cases the ankle-jerk was diminished or absent in 7 cases, and present or exaggerated in 5 cases. The knee-jerk was present or exaggerated in 7 cases and diminished or absent in 5 cases.

The superficial reflexes are often of additional value in indicating the level of the lesion. The plantar reflexes are either normal or result in no response. The latter may occur in the presence of cutaneous analgesia or of flaccidity of the muscles of the posterior compartment of the leg. The cremasteric reflex may be absent if the tumour affects caudal roots at the level of the second lumbar nerve-root. Thus the cremasteric reflex may help to localise the lesion at a higher level than is indicated by other symptoms or physical signs. In some of the cases reported the lower abdominal reflexes have been absent, but in view of the obvious difficulty of assessing the value of the loss of these reflexes on both sides especially in females and elderly patients it is difficult to draw any conclusion from this finding. Absence of the reflex on one side, however, may suggest that the lesion has reached a higher level than is suspected from the other symptoms and physical signs and thus be of considerable value in estimating the probable results of treatment.

In connection with the reflexes, as with motor and sensory functions, the roots both above and below the level of that involved may be unaffected, even though from the position of the tumour it might be expected that they would be. It is apparent, however, that the roots involved indicate that the upper level of the tumour has at least reached the level at which they leave the spinal canal, but that they may fail to give any indication of either its upper or lower limit because of the perpendicular course of the roots. In approximately half of the cases the upper limit of involvement of the reflexes gives an

indication of the level of the upper limit of the tumour, while in the remaining half the level of involvement of the reflex arc indicates a lower level, or the reflexes are entirely unaffected.

(5) *Trophic Disturbances.*—Trophic disturbances are present fairly often in the more advanced cases of caudal tumours. Atrophy of the affected muscles and the electrical reactions present have been referred to above. Congestion, dampness or coldness of one or both feet is sometimes present. Occasionally there is œdema of one or both feet. In one case there was massive, hard œdema extending up to a level just below the knees and presenting all the characteristics of the trophœdema usually associated with Milroy's disease.

Trophic sores may appear in the lumbosacral region or over the buttocks, and less often over the great trochanter or the heels. Such trophic sores are, however, present only in the few cases confined to bed in an advanced stage.

Œdema of the lower limbs was noted in two of Parker's cases, and Bailey has referred to a case described by Schmoll in which the œdema was so extensive that a laparotomy was performed to exclude a tumour pressing on the inferior vena cava.

(6) *X-ray Examination.*—X-ray examination of the lumbosacral spine is an essential part of the routine examination of all cases of suspected caudal tumour. In the majority of cases no abnormality whatever is detected. Occasionally evidence of absorption of the laminæ or of the bodies of the vertebræ is found and may indicate the effects of local pressure by a tumour within the spinal canal. In one case reported by Wolfsohn and Morrissey[12] there was cyst-like absorption of the posterior half of the bodies of the fourth and fifth lumbar vertebræ, while the laminæ were thin and hazy. At operation a large solid fibroma was found filling most of the spinal canal dorsal to the bodies of the fourth and fifth lumbar vertebræ. Occasionally evidence of malignant disease may be found in the bodies of the lumbar or sacral vertebræ.

Most commonly changes due to arthritis are found in the lumbosacral vertebræ. In some cases they may be enough to account for the symptoms, as in the group of cases described by Bailey and Casamajor[29], but more often they render the final diagnosis more difficult. In one of Davies'[30] cases the X-ray findings characteristic of osteoarthritis added much to the difficulty of diagnosing a caudal tumour. In one of Elsberg's cases extensive arthritic changes were present in all the lumbar vertebræ, while in another there was spondylitis of the second and third lumbar vertebræ together with atrophy and thinning of the lumbar and upper sacral vertebræ. Apart from the increased difficulty in diagnosis which results from such findings, it is probable that if the causes of arthritis are active in the patient the condition is particularly apt to appear in a part whose resistance is lowered by the presence of a caudal tumour.

Congenital defects of the sacral spine may be found and spina bifida in particular. First described by Fuchs[31,32] under the name of myelodysplasia and subsequently elaborated by Lewandowsky[33], Peritz[34], Saenger[35] and

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Spiller[36], these defects are of considerable importance in diagnosis. In some cases the interpretation of X-ray findings may be exceedingly difficult. In one of Parker's[3] cases there appeared to be an area of decreased density in the first and second sacral vertebræ, possibly the result of pressure by a tumour, but at operation it was found that the changes were due to a spina bifida occulta involving the arches of the first and second sacral vertebræ. In one of Elsberg's cases a caudal tumour was associated with a defect in the middle line involving the first and second sacral vertebræ, which afterwards proved to be a spina bifida. Cushing and Ayer[20] reported an interesting case of anterior spina bifida associated with a caudal tumour, in which there was great difficulty in deciding what condition was responsible for the patient's symptoms and physical signs.

The more special methods of examination involving the use of X-rays are discussed later.

(7) *The Results of Lumbar Puncture.*—The clinical findings on lumbar puncture in the classical position fall into three main groups, viz., (*a*) failure to obtain fluid ; (*b*) variations in the loculation syndrome ; and (*c*) misleading findings.

A ' dry tap ' resulted in 33 per cent. of the cases reported in the literature and in 6 out of Ott and Adson's 12 cases. In some cases no fluid is obtained on repeated punctures at the usual level and in other positions. The reason for the dry tap varies in different cases. In 60 per cent. of the cases of dry tap there was present in the spinal canal a ' giant tumour ' of the type described by Collins and Elsberg[37]. In the remaining 40 per cent. the dry tap is the result of accidental insertion of the needle at the level occupied by the tumour. In the latter group of cases fluid is obtained by puncturing space by space above and below the site of the original puncture. Repeated dry taps by a skilled operator may be regarded as evidence that the spinal canal is probably occupied by a tumour ; and occasionally the finding of fluid one or two spaces above the site of the original puncture is of value in fixing the upper level of the tumour.

The characteristics of the cerebrospinal fluid obtained below the lesion vary considerably. In only 13 per cent. of the cases recorded was it found to be normal. A normal cerebrospinal fluid is not necessarily associated with absence of definite physical signs confirming the presence of a caudal tumour. It is, however, the slight abnormalities in the cerebrospinal fluid of cases with an indefinite onset which help to confirm the presence of a caudal lesion. Recently, Elsberg and Constable[16] have expressed the opinion that with both extradural and intradural tumours of the cauda equina, whether a subarachnoid block can be demonstrated or not, the total protein of the cerebrospinal fluid is always found well above the normal. Nevertheless, their conclusion that a diagnosis of neoplasm in the lowermost part of the vertebral canal is not justified if the total protein is found to be within normal limits on lumbar puncture appears to be too dogmatic.

The fluid was yellow in 63 per cent. of the cases in which fluid was obtained by lumbar puncture from below the level of the tumour, while in two cases yellow fluid was found in an encapsulated space. The fluid varied in colour from a faint yellow colour to a deep golden yellow. In 29 per cent. the total protein or the globulin alone was increased without any change in the colour of the cerebrospinal fluid. In both the yellow and the colourless fluids the cells were within normal limits.

Misleading changes were found in a small proportion of cases. Occasionally 6 or 8 cells were present and less commonly as many as 25 or 28. In one case reported pus cells were present in the fluid for no obvious reason and in another 300 cells were present.

In one of Bennett's[19] cases there was removed in the lumen of the needle a small piece of tissue which on microscopical examination proved to be a piece of tumour. In some cases, even though no fluid was obtained, the impression was gained that the needle was passing through soft friable material.

In view of suggestions which have been made as to the source of the changes in the cerebrospinal fluid the cases in the literature were reviewed to determine the presence of any relationship between the nature of the tumour and its position and the changes in the fluid. There appears to be no relation between the type of tumour and the changes in the fluid. In 87 per cent. of cases in which marked changes are present the tumour is situated within the dural sac, but in the majority of cases in which the fluid is normal the tumour is also intradural.

In seven cases out of nine in which the examination was made pronounced changes have been found in the fluid above the level of the tumour. Such changes have been recorded on deliberate puncture above the tumour, on accidental puncture, at operation and on examination of the fluid from the cisterna magna. In the case of accidental puncture above the level of the tumour the changes may lead to the belief that the tumour is above the level at which the puncture is made, and in two cases reported by Cushing and Ayer[20] resulted in exploration of the spinal cord instead of the cauda equina. An equal amount of protein above and below the tumour has been reported in a case by Marie, Foix and Robert[38], while in a further case described by Marie, Foix and Bouttier[39] it was suggested that the fluid above the tumour was abnormal. Ayer discussed the significance of different types of abnormal fluid from both the cisterna magna and the lumbar sac, and concluded that generally the increase in protein above the tumour was less than that below. No attention was, however, paid to the occurrence of an abnormal fluid above a tumour of the cauda equina until Cushing and Ayer's report of five cases in 1923. In Raven's[40,41] analysis of a series of cases of spinal tumour, a positive Nonne phase I reaction was described in one cerebrospinal fluid evidently obtained above a tumour of the cauda equina. The changes in the cerebrospinal fluid above the tumour in Cushing and Ayer's cases were similar to those below but less pronounced. In all five cases the fluid was yellow and the

protein in excess. Xanthochromia in the cisternal fluid was noted in one case, and also in a case reported by Hammes[26].

Opinons differ as to the cause of the changes in the cerebrospinal fluid above and below the tumour. Ayer agrees with Sprunt and Walker's[42] opinion as to the significance of xanthochromia and regards the changes above, as below, to transudation from engorged veins, while Cushing believes them to be due to direct transudation from the tumour itself. In both cases it is believed that the fluid below becomes entrapped and thus concentrated, while that above escapes and becomes diluted. Cushing cites in support of his explanation the fact that in all of his cases the tumour was intradural. A review of the cases of caudal tumour described, however, shows that in 13 per cent. of those in which the fluid was abnormal the tumour was situated extradurally, and that in practically all the cases in which the fluid was normal the tumour was situated intradurally.

VI.—THE LOCALISATION OF CAUDAL TUMOURS.

The localisation of tumours of the cauda equina is often very difficult. The perpendicular course of the caudal roots within the spinal canal is the chief source of difficulty, for it is possible for a root to be involved in any position from the end of the spinal cord to its exit from the spinal canal. Hence from the signs of involvement of individual nerve roots it is impossible to determine the lower limit of the tumour. In many cases, however, it is possible from the study of signs of involvement of individual nerve roots to gain some idea of the position of the upper limit of the tumour. The following clinical features, when considered together, may give information of value : (1) pain in the back ; (2) segmental pain in the lower limbs ; (3) the upper limit of changes in sensation ; (4) the level to which the reflexes are involved ; (5) the level to which the motor functions are affected ; and (6) the results of lumbar puncture. Cases occur in which it is impossible to localise the upper limit of the tumour. Each case has to be considered carefully on its merits, and it is only by so doing and by applying the results of past experience that some idea may be gained of the position, nature and extent of the tumour.

Pain in the back has been mentioned by Spiller[15] as a clinical factor of value in some cases. It has been shown, however, that the inaccuracy with which pain in this region is often described by the patient is usually a contra-indication to the use of the symptom in localisation. Cases occur, however, in which the pain is described with sufficient accuracy to indicate that the lesion has at least reached a certain level.

Nerve-root pains in the lower limbs would appear to be of greater value. A study of cases reported, however, shows that in only 10 per cent. of cases does the level of the root pain indicate the upper limit of the lesion. In rare cases it is of particular value. In a case reported by Lind and Lundstein[22] the pain was in the distribution of the second lumbar root and aton operati

a neurofibroma was found growing from the second lumbar posterior root. Localisation was possible in a similar way in cases described by Davies[30] and Elsberg[2].

The level to which the different types of sensation are involved is occasionally of value, but again fails very often partly because anæsthesia and analgesia to a level corresponding to the upper limit of the lesion are usually a late manifestation. It has been shown that in approximately one half of the cases this sign alone indicates the upper limit of the lesion but that in the other half the lesion is usually at a higher level.

The level of motor involvement is as a rule of less value because of the difficulty of determining the upper level of muscular weakness. If a lumbar puncture results in a dry tap, a series of lumbar punctures from below up may eventually reveal the presence of fluid and show that the upper level of the lesion is below the level at which it was obtained.

It is found, however, that the best clinical method of determining the upper limit of the lesion is to take the uppermost of the upper levels of involvement as shown by the subjective and objective sensory changes, the changes in motor function and the variations in the reflexes. The latter are often helpful especially when one cremasteric reflex is absent and the other present. Occasionally a clue can be obtained to the lateral and antero-posterior position of the tumour. A preponderance of physical signs and even of subjective sensations on one side suggests that the tumour is placed rather to that side; while a preponderance of sensory signs over motor or vice versa suggests that the tumour is placed either behind or in front of the roots of the cauda equina. No general deductions can be made on such evidence though it is sometimes found to be useful in individual cases. Elsberg[2] has, however, pointed out that large soft tumours of the cauda equina which fill up the spinal canal are apt to produce largely motor symptoms and signs.

In early cases the only evidence suggesting the presence of an intraspinal tumour is the abnormality of the cerebrospinal fluid. A comparison of the fluids obtained by one or more lumbar punctures with that obtained by cisternal puncture sometimes reveals differences in the fluids at the different levels. An examination of the fluid from different levels above the tumour may reveal less colour or less protein at the higher levels. When lumbar puncture is performed below the tumour, the pressure of the fluid and the variations in the pressure with respiration and on superficial and deep jugular compression are less than those above. Ayer points out that if normal cisterno-lumbar pressure relations are obtained the tumour is probably below the site of lumbar puncture. Ayer and Mixter in particular have combined lumbar and cisternal puncture and consider that lumbar puncture alone is of little localising value. Cushing and Ayer[30] have used the following method to determine the presence or absence of a tumour in the lower part of the sacral canal. A needle connected with a manometer is inserted above the level of the suspected tumour and at the

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same time 70 c.c. of saline injected into the epidural space through the sacral notch. Normally this injection produces a continuous rise of fluid in the manometer but in the presence of a tumour below the level of the upper needle this rise is very slight or absent.

A manœuvre suggested by Viets[28] may help to determine the upper limit of the lesion. If lumbar puncture has been performed below the level of the block as indicated by the changes in the fluid, the comparison of the lumbar and the cisternal fluids and of the lumbar-cisternal pressure, and then the fluid is drained from below the block, jugular compression may produce intense pain in the segmental area corresponding to the uppermost root affected by the tumour. This manœuvre has been reported in only one case, in which jugular compression after drainage of the cerebrospinal fluid from below the block produced pain in the upper part of the front of the thigh, disappearing on removal of the pressure. In this case a neurofibroma was found attached to the second right lumbar nerve root. Viets explains that by draining the fluid below the block the water-bed is removed and the collection of fluid above then acts as a buffer through which jugular compression exerts pressure on the tumour and produces traction on the nerve root.

Certain French workers have injected air corresponding to the amount of fluid removed and found that even when the block is only partial pain occurs in a nerve root area corresponding to the level of the tumour.

In some cases an accurate localisation may be possible only by the use of lipiodol according to the method introduced by Sicard. In the case of cisternal puncture lipiodol of high specific gravity is introduced and the level at which it is held up determined by X-ray examination. When a lumbar puncture is done below the level of the tumour lipiodol of low specific gravity may be introduced there and the lower limit of the tumour thus defined. Viets[28] has pointed out that shifting of the lipiodol shadow with changes in the position of the tumour is a characteristic feature of a caudal tumour free within the spinal canal and of particular importance in the differentiation of such a tumour from a tumour of the spinal cord or from one attached to the spinal meninges.

Recently Elsberg and Cramer[43] have pointed out that severe root pains and disturbances of the bladder have continued for some days and inflammatory changes have been found in the roots of the cauda equina after the use of iodized oil. In attempting to avoid these ill-effects they applied the method of multiple lumbar punctures used by Stookey and Klenke[44] to the localisation of tumours of the cauda equina. They found that it was possible to localise a tumour of the cauda equina and especially its upper and lower limits with some degree of certainty by this method. A failure to obtain fluid at any puncture indicated the presence of a giant tumour, while the demonstration of a complete or partial subarachnoid block by means of pressure readings at one level and not at another showed that the tumour was situated between the two levels, and the absence of a block at both the first and the fifth lumbar interspaces that it was probably situated in the sacral part of the lumbar canal.

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VII.—DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

The general features upon which the diagnosis of a caudal tumour is based may be readily understood from the description of the symptoms and physical signs given above. This applies, however, to a more or less well-developed condition. Generally it may be accepted that if alterations in the subjective or objective sensory functions, motor functions or in the reflexes occur bilaterally the lesion responsible is in the spinal column or spinal canal if a lesion of the peripheral nerves can be excluded. Further investigations along the lines suggested above are then necessary to localise the lesion and, if it is found to be within the spinal canal itself, to determine its nature and position. Bilateral pain in the buttocks or in the distribution of the sciatic nerves, loss of both ankle jerks, wasting and sometimes fibrillation of muscles in both lower limbs and less often bilateral objective sensory changes are enough to suggest that the lesion is in the spinal canal or in the spine itself. It cannot be too strongly urged, however, that bilateral changes in any one of these groups alone is enough to give rise to the suspicion that a caudal tumour is present.

In the majority of cases, however, the symptoms and signs in the early stages are entirely unilateral. Frequently physical signs are entirely absent. Reference to the description of the initial symptoms shows that in over 90 per cent. of cases pain either in the back or referred to a root area in one or both lower limbs is the symptom which appears first. It is not always constant and may disappear only to reappear either in the same or in the other lower limb. In view of the gravity of caudal tumours diagnosed at a late stage it is important in all cases showing such symptoms to examine the cerebrospinal fluid in detail. In the event of a repeated dry tap or of changes in the fluid itself it is then necessary to apply the other methods described to confirm the presence or absence of a caudal lesion. In this connection the fact that minor changes in the cerebrospinal fluid sometimes occur in true sciatica does not in any way contra-indicate further investigation

The sources of error in the diagnosis of caudal tumours in the early stages and the conditions from which they must be distinguished are numerous.

The differential diagnosis from unilateral or bilateral sciatic neuritis depends upon the thoroughness of the investigation, though in the latter case the chances are strong that the lesion is in the pelvis, spinal column or spinal canal. It must be recognised that many of the signs and symptoms of sciatic neuritis may be present with a tumour of the cauda equina. Pressure on the sciatic nerve in any part of its course may result in pain either locally or in the distribution of the sciatic nerve, there may be difficulty in extending the knee joint beyond ninety degrees without producing pain, Lasegue's sign may be present and the muscles may be tender to pressure. The presence of such findings may confirm an erroneous opinion that a condition with unilateral pain is sciatic neuritis and not a caudal lesion.

In multiple neuritis the widespread distribution of the disease, the characteristic sensory changes, the variations in the reflexes, the symmetrical

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character of the clinical features and in most cases the absence of involvement of the sphincters serve to differentiate the two conditions. The form of amyotrophic lateral sclerosis in which the lower motor lesion affects the upper sacral segments sometimes causes confusion, but signs of the spastic element of the disease elsewhere or in the lower limbs usually suggest the nature of the condition. In both cases, whenever the diagnosis is in doubt, it is necessary to continue the special investigations until a caudal lesion is excluded.

Myositis, fibrositis, lumbago and other conditions which may bring the patient to an orthopædic surgeon rather than to a neurologist may cause difficulty, again because of the ease with which the clinical features are ascribed to the muscles or fibrous tissues of the back. Bennett[19] has drawn attention particularly to spasm of the erector spinæ muscles and of the hamstrings together with rigidity of the lumbosacral portion of the spinal column in the early stages of caudal tumours.

Tumours within the pelvis must always be excluded especially when unilateral or bilateral pain is present. For this purpose a complete examination and exploration of the pelvis is often necessary.

Lesions of the spinal column itself often present the most serious problems in diagnosis. Spinal caries of tuberculous origin is stressed by Péron[14] as of particular importance in differential diagnosis, while secondary malignant disease of the vertebræ must also be considered. Both tuberculous and malignant conditions of the spinal column may reveal themselves initially as lesions of the cauda equina, but they are as a rule readily identified on X-ray examination and on consideration of the possible sites of tumours elsewhere.

Hypertrophic arthritis of the lumbosacral spine is readily identified on X-ray examination, which must be applied as a routine method of examination in these cases. Sacralisation of the fifth lumbar vertebra may sometimes result in damage to the fifth lumbar root on one or both sides and so produce pain, anæsthesia and motor weakness. Bailey and Casamajor have drawn attention to osteo-arthritis of the spine as a cause of compression of the spinal cord and its roots and Sachs and Fraenkel[45] in particular have described a case in which the clinical features resembled very closely those of a caudal tumour. It must not be forgotten, however, that the presence of these conditions does not necessarily exclude the diagnosis of a caudal tumour. The two conditions may quite well be present together and in that case a useful method of differentiating the lesion at the lower level from that at the higher level as the cause of the symptoms is to inject epidurally a solution of novocaine and to note the effect of the injection upon the pain.

The group of conditions collected by Fuchs[31,32] under the name of myelodysplasia is often a source of difficulty and more especially the various modifications of spina bifida associated with some involvement of the caudal roots or even of the conus. In his original paper Fuchs described the clinical features as weakness of the sphincters and especially enuresis nocturna persisting after puberty, syndactylism, disturbances of sensation chiefly thermal and not

strictly segmental, particularly in the feet and toes, a defect in the sacral canal recognised on X-ray examination, anomalies in the cutaneous and tendon reflexes, and defects in the feet sometimes with weakness of the peroneal muscles and trophic and vasomotor disturbances. Mattanschek and Fuchs[31] found similar anomalies in 87 per cent. of adults with enuresis alone, and Peritz and Sachs in 68 per cent., while Saenger[35] found only one adult with myelodysplasia among patients with enuresis, and Vorkastner quoted Lewandowsky as stating that the conditions were merely associated. The absence of pain in such conditions, the long duration of the symptoms and the recognition of a defect in the sacral canal on X-ray examination often serve to differentiate the condition from a caudal tumour. It is obvious, however, that on these considerations the differentiation may fail completely. Spiller[36] has drawn attention to the fact that the condition may be entirely symptomless, and symptoms and physical signs appear only in adult life after violent exercise. In any case, just as with hypertrophic arthritis, the presence of spina bifida occulta does not exclude a diagnosis of caudal tumour. The two conditions were associated in cases reported by Parker[3], Elsberg[2] and others. In one of Cushing and Ayer's[20] cases the diagnosis was for long in doubt because of the presence of an anterior spina bifida, a much rarer form of the condition. In addition, the thinning of the laminæ of the vertebral arch or of the bodies of the vertebræ by pressure from a tumour may give the impression on X-ray examination that a spina bifida is present.

Neuritis of the cauda equina, cases of which have been described by Oppenheim[46], by Kennedy, Elsberg and Lambert[47], and by Reynolds[48], is sometimes a source of difficulty. Such cases have usually come to operation and the diagnosis been made on exposure of the caudal roots. In all of the cases reported and in Parker's[3] case the diagnosis was made at operation and the caudal roots were found to be swollen, congested and matted together. Elsberg and Constable[16] recently contrasted the clinical features of a series of cases of neuritis of the cauda equina and of tumours of the cauda equina and dealt with the differential diagnosis in detail.

Meningoradiculitis involving the caudal roots, tuberculous or syphilitic in origin, and especially the sacral form of tabes dorsalis are as a rule readily excluded on examination of the patient, the cerebrospinal fluid and a blood Wassermann reaction. In connection with the former, however, it is important to note that in a few cases of caudal tumours the cell content of the cerebrospinal fluid has been increased for no obvious reason.

The differentiation of pure caudal tumours from those which involve the conus either primarily or secondarily is of particular importance when planning treatment and attempting to make a prognosis. A study of caudal tumours shows that those in which sudden and serious involvement of the sphincters has occurred either early or at any stage in the clinical course are found either at operation or at autopsy to have the conus implicated.

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It may be extremely difficult to differentiate conditions involving the epiconus from caudal tumours. The epiconus syndrome as described by Minor[49] and revised by Alpers[50] consists of both positive and negative symptoms, the latter being quite as important as the former. The former are signs of involvement of the fifth lumbar and first and second sacral roots with absence of the ankle-jerk, while the latter consist of preservation of the knee-jerks and normal sphincters. While the outstanding feature is involvement of the peroneal muscles on both sides, the glutæi, hamstrings and extensors of the toes may be affected as well. In fact Seletzky[51] has suggested two types of epiconus syndromes, the first characterised by involvement of the glutæus maximus being due to a lesion of the lower part of the epiconus, and the second by peroneal paralysis due to a lesion of the upper part of the epiconus. Cases have been described in injury of the spinal cord by Minor and by Weisenburg[52], in poliomyelitis by Minor[53] and by Spiller[54], in spina bifida occulta by Tutyshkin[55], in tabes dorsalis by Alpers, as the result of an infiltrating tumour by Ornstein[56], and also by Parker[3] and Levison[57]. It may occur as a result of traumatic myelitis, of myelitis due to other conditions, and of myelomalacia from vascular degeneration and thrombosis. The absence of pain is rarely of value in differentiation especially as pain may occur in an anæsthetic area with an epiconus lesion. The order in which the symptoms and physical signs appear together with a strong tendency to asymmetry in caudal tumours is often of value in differential diagnosis.

It might not be thought that tumours of the spinal cord itself would present any difficulties in diagnosis. Sargent[58] has, however, drawn attention to the occurrence of what appear to be root pains in the lower limbs in some cases of spinal cord tumour, possibly as a result of pressure upon selected pain fibres in the lateral column of the spinal cord. In such cases, however, a complete neurological examination is usually enough to enable one to decide that the symptoms are those of a lesion at a higher level than that of a caudal tumour.

VIII.—DISCUSSION.

A review of the clinical features of tumours of the cauda equina reveals many points which are of value in the study of an individual case, and shows that certain general principles often applied to the study of caudal tumours have no actual foundation in experience.

The symptomatology of caudal tumours cannot be assumed from the anatomical and physiological functions of the parts which form the cauda equina. Nor can they be assumed from a study of the traumatic lesions of the cauda equina. It is evident that the application of the general principles obtained by a study of the anatomical and physiological features and of traumatic lesions will result in the diagnosis of caudal tumours in many cases only when treatment is of no avail. If the object of the investigation of the patient is to be the successful treatment of the condition from which he suffers, which is

possible only if the condition is diagnosed in its early stages, some more satisfactory basis must be found for the investigation of such lesions.

It is necessary to separate the symptomatology of tumours of the cauda equina itself from those due to involvement of the conus in addition to the cauda equina. In order to do this it is essential to define pure caudal tumours as those entirely below the lower border of the second lumbar vertebra. Having done this the significance of disturbances of the sphincters is at once apparent. The late and gradual appearance of sphincter disturbances then becomes apparent as a feature of caudal tumours, whereas the rapid appearance of sphincter disturbances at an early stage or at any part of the clinical course would appear to indicate that a purely caudal tumour has become complicated by involvement of the conus.

The habits of a caudal tumour in regard to its symptoms, physical signs and clinical course cannot be compared with the clinical picture produced by experimental or accidental injury to the cauda equina. A tumour in this region, even when very extensive, appears to follow no general rules in regard to the structures it involves. Hence some structures are affected and others unaffected for no obvious reason. The symptoms and the physical signs vary in consequence.

The importance of the recognition of the initial symptoms and of the value of pain as a symptom has been established. The influence of the recumbent position and of movement on the pain in the early stages is an important diagnostic feature. Pain in the back would appear to be of greater value in drawing attention to the condition than pain confined to a segmental area in one lower limb. This finding has the obvious corollary that cases of chronic pain in the back and in the sacral region must be investigated as carefully for evidence of a cauda equina tumour as are those of sciatica. Another point of importance which has emerged from this study is the frequency of intermissions in the pain before the appearance of physical signs upon which a tumour of the cauda equina might be suspected on clinical evidence alone. The intermissions appear to vary in duration according to the rate of involvement of the cauda equina, lasting from three months to as long as seven years. The second attack of pain may not be in the same position as during the original attack. One attack of sciatica followed by a second attack in the other lower limb after an interval of one or two years does not necessarily mean two attacks of sciatica ; it may be an indication of the bilateral distribution of symptoms upon which the presence of a caudal tumour is usually suspected.

It is evident, however, that in the early stage and especially when pain is the most prominent symptom, a diagnosis of a caudal tumour cannot be made even on a complete neurological examination. It is necessary to introduce special methods of investigation, of which lumbar puncture appears to be the most important. The need of attempting to obtain and examine the cerebrospinal fluid in every case of persistent pain in the back, in the sacral region, in a segmental area or even in a part of a segmental area is established. The

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importance of a repeated dry tap is evident and the significance of slight changes in the cerebrospinal fluid must be emphasised. In the presence of such minor indications the value of X-ray examination of the spine and of further attempts to localise the lesion by means of multiple lumbar punctures or lipiodol cannot be under-estimated. The significance of pain during the 'période de début' is interpreted erroneously in nine cases out of ten when the investigation is confined to the usual clinical procedures.

When physical signs appear they are often in the first place asymmetrical. Any evidence of the bilateral distribution of symptoms and physical signs, whether it be in regard to subjective sensations, objective sensory changes, changes in the reflexes, or alterations in motor function, should and in fact usually does lead to the suspicion that a lesion exists at the level of the exit of the nerves from the spinal canal or at a higher level. Here again detailed investigation is essential. The importance of studying such changes on first principles as evidence of involvement of individual nerve roots must be recognised so that the nature and position of the lesion may be determined. Evidence of involvement of some caudal roots and of the sparing of others is particularly valuable in the diagnosis of caudal tumours at an early stage.

It may be accepted that the physicial signs of caudal tumours at the stage when a diagnosis may be made on clinical findings alone do not necessarily follow any definite type. It is only in the later stages that the clinical picture can be said to conform to a type or to resemble in any way the clinical descriptions.

The importance of abnormalities of the cerebrospinal fluid has been confirmed and in addition the source of error due to changes in the fluid above the tumour has been emphasised.

Accurate localisation of the tumour is necessary for the most successful treatment. The upper limit can probably be determined more or less accurately in some cases by a careful study of the symptoms and physical signs. In the majority of cases in which physical signs are present this method results in an approximate localisation. The lower limit can sometimes be determined by multiple lumbar punctures or by the introduction of lipiodol of low specific gravity below the tumour. The lateral position of the tumour may sometimes be assumed from the more or less unilateral distribution of the symptoms and physical signs, and the antero-posterior position from the relative preponderance of symptoms and signs of involvement of motor or of sensory roots. The latter generalisation is, however, subject to the difficulty that giant tumours of the cauda equina may involve motor roots to the entire exclusion of sensory roots.

The differential diagnosis of caudal tumours depends upon a careful and detailed investigation of the patient. Occasionally sources of difficulty arise, as in the not uncommon association of hypertrophic arthritis of the spine or of spina bifida occulta with the lesions under consideration. In such cases the

chances of confusion are lessened by assuming that the clinical picture is the result of the more serious condition and carrying the investigation of the patient further until this is proved or disproved.

IX.—SUMMARY AND CONCLUSIONS.

1. This paper is based on a review of the clinical features of cases of tumour of the cauda equina, and confined to the study of those in which the presence, nature and position of the tumour have been established either at operation or at autopsy.

2. The successful study of tumours of the cauda equina depends upon the separation of those in which the cauda equina alone is involved from those in which the conus and the structures above it are involved in addition to the cauda equina.

3. The various methods of approach to the study of the clinical features of tumours of the cauda equina are considered in detail. With the exception of the 'hemicaudal' and 'radicular' syndromes of Roussy and Lhermitte none of them appears to offer a satisfactory basis for the study of such lesions. The only feasible method appears to be the study of the clinical features on the basis of the evidence of involvement of individual nerve-roots and the interpretation of the findings so made in the light of experience of the clinical features of tumours of the cauda equina.

4. The clinical course of tumours of the cauda equina is reviewed, the results of treatment at different stages studied and the conclusion reached that successful treatment must depend upon the diagnosis of the condition in its early stages.

5. The importance of pain as the initial symptom is confirmed, and emphasis laid on the fact that pain in the back may be a more common initial symptom than segmental pain in the lower limb.

6. The clinical features of tumours of the cauda equina are discussed in detail and the conclusion reached that it is only in the well-developed case that any particular type of clinical picture is found. Important points would, however, appear to be the asymmetrical character of the clinical findings and the appearance of changes bilaterally in one or more of subjective sensations, objective sensations, motor functions and reflexes.

7. The stage of onset is the stage of diagnostic errors. If investigation is confined to ordinary clinical methods at this stage such a position is inevitable. Consequently special methods of examination and especially a detailed examination of the cerebrospinal fluid are essential.

8. When one or more physical signs are established the bilateral distribution of any of them is suggestive and further investigations may then be necessary to confirm the diagnosis and to localise the lesion.

9. The tendency of tumours of the cauda equina to affect the nerve roots in a haphazard manner is mentioned and emphasis laid upon the effect of this

characteristic in making it impossible to study such tumours according to the clinical descriptions of traumatic lesions of the cauda equina or according to recognised syndromes.

10. The delay in investigation and the postponement of surgical treatment until a clearcut type of clinical picture is present means in most cases waiting until the condition is beyond the resources of treatment.

11. The various conditions with which tumours of the cauda equina may be confused are mentioned and emphasis laid upon possible sources of confusion in differential diagnosis.

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FURTHER NOTES ON EXAMINATION OF CEREBRO-SPINAL FLUID BY ULTRA-VIOLET LIGHT.

By

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In a previous paper* I described some observations on the absorption of ultra-violet light by cerebrospinal fluid. In the course of these observations it was noticed that there was a certain amount of general absorption of the light waves varying with different fluids and that the most striking of these results occurred in cases of tuberculous meningitis.

These results were obtained with a primitive apparatus and were rightly criticised on the ground of insufficient standardisation; so a further series of observations has been made during the past year to standardise results as far as is possible under the conditions in which the work has had to be carried out, conditions hampered by my own lack of training in research work and the unavoidable spasmodic nature of the observations.

My previous observations were made with a copper arc since I found the tungsten arc very expensive to work with, as the metal burned away so quickly; but Mr. Clarke, of the Physics Department of the Sheffield University, to whom I am indebted for very valuable assistance, suggested that the tungsten spectrum was a much more reliable band to work with and that most of the recent observation on ultra-violet light had been done with this spectrum, and I therefore decided to use this metal for the next series. I modified the apparatus so that instead of using a direct current to energise the copper rods, I adapted an old X-ray 32-inch induction coil with a condenser of ·005 microfarad capacity in parallel with the spark. This apparatus, taking a primary current of 25 amps. at 250 volts, produced a fat spark across the tungsten rods when the latter were separated by a gap of 3 millimetres, which gap was used throughout the experiments. The rest of the apparatus, which was set up on a home-made optical bench, consisted of a Hilgar spectrometer of low dispersion, a quartz lens to focus the rays and a suitable screen to hold the quartz cell containing the spinal fluid. These cells were made of two plane parallel faces of quartz 4·5 cm. in diameter cemented to sections of glass tubing.

Observations were first carried out to determine the best results from a photographic point of view and it was found these were given by an exposure of

* See this Journal, October 1929, vol. x, p. 97.

5 seconds, using Wellington panchromatic plates, which were afterwards developed by time and temperature method, using standard rytol.

One question raised by the previous paper was the effect on the spectrogram of varying thicknesses of fluid and a preliminary series of observations was made to ascertain what such difference might be. Cells of ·25, ·5 and 1 cm. were made and it was found that unless the fluid was very turbid spectrograms taken through these varying thicknesses of fluid showed remarkably little difference; after a number of tests a cell with a depth of ·5 cm. was chosen as the standard and all spectrograms were taken through this thickness of fluid, unless otherwise stated.

Fig. 1 shows the general lay-out of the apparatus.

FIG. 1.

Fig. 2 shows the normal spectrogram of tungsten produced under the above conditions. It is, of course, sparking in air which itself absorbs a certain amount of the ultra-violet waves and an attempt was made to obtain an *in vacuo* spectrum.

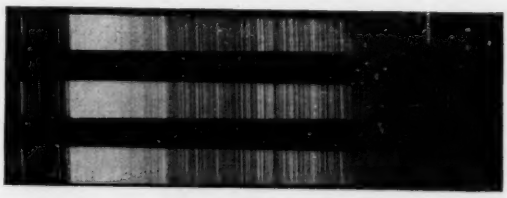

FIG. 2.

Fig. 3 shows such an attempt. So far as I could see there was no difference between the two, and the added difficulty was such that I did not consider it necessary to carry out any further *in vacuo* observations, particularly as absorption in the fluids concerned was found well before the end of the spectrum

taken in air. (It will be noted that the last spectrum is one of copper and not tungsten.)

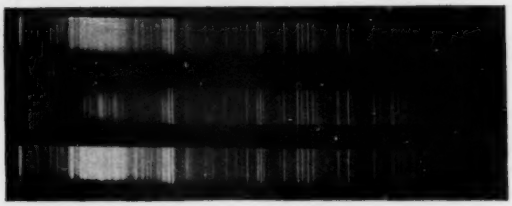

FIG. 3.

Fig. 4 shows the spectrogram of spinal fluid photographed through thicknesses of ·25, ·5 and 1 cm. respectively, as mentioned above, and it will be seen that the difference is quite a small one.

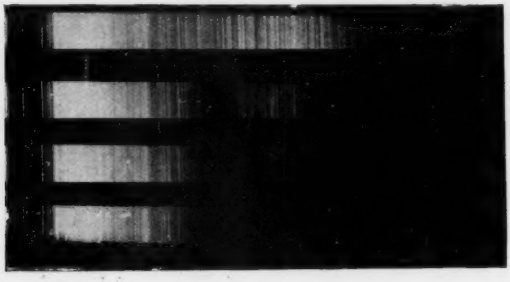

FIG. 4.

Fig. 5 is the spectrum of tungsten with some of the more easily identified wave-lengths marked. It will be noticed that these wave-lengths vary in value from 4348 a.u. somewhere near the blue of the visible spectrum, to 2256 a.u. well down in the ultra-violet band.

FIG. 5.

With this rather long preface I will now very briefly describe the findings in the second series of observations that have been made, and I would like to quote the last paragraph of my previous paper : " The conditions I have cited give spectrograms which appear more or less constant in type but on what they depend at present I cannot say. I am quite sure they do not depend on the number of cells present in the fluids nor do the variations in the protein constituents appear to bear any fixed relationship. The only definite statement possible is that absorption seems to take place in toxi-infective conditions and varies directly with the severity of this process ; the nearer a fatal termination the more complete the absorption."

It is, of course, perfectly clear that changes such as I am describing in one tissue (spinal fluid) cannot carry a diagnostic label ; our labels are too gross for such minuteness. For instance the label ' general paralysis ' conveys to our minds a more or less definite clinical picture ; but the picture is really a moving one inasmuch as we envisage a series of events occurring in time, whereas an examination such as we are making can only be the results of certain metabolic accidents at a given moment of time and such results as I have obtained must vary at different stages within the range of one disease-process.

FIG. 6.

It is manifestly absurd to think, for instance, that every case of general paralysis must give the so-called paretic gold curve in the Lange test since the factors on which this test depends are probably only progressively developed. The results I have obtained spectrographically, if they have any value at all, can only suggest that certain tendencies are at work and can never have the same evidential value for a given instant as say, an X-ray picture of a fractured bone.

With this fact clearly stated I may be allowed to use the clinical labels for convenience and confine myself to the results I have obtained in cases of meningitis.

Fig. 6 shows the amount of absorption by normal spinal fluid, this fluid having been obtained from a perfectly healthy volunteer from the Labour Exchange, and it will be observed that absorption only begins at a wave length of 2397 a.u.

Fig. 7 gives the characteristic spectrogram from a case of tuberculous meningitis. It can be seen that a very considerable amount of ultra-violet light is absorbed, the absorption commencing about a wave-length value of 3051 a.u. I will quote the pathological report on this particular fluid :—

Clear fluid containing a ' spider ' clot.
Leucocytes ... ... 80 per c.mm.
Protein increased.
Chlorides ... ... ·75 per cent.
Gold test 001223342

Autopsy (two days later). Many tubercles on vertex of the brain ; much basal exudate. Miliary tubercles in spleen and lungs.

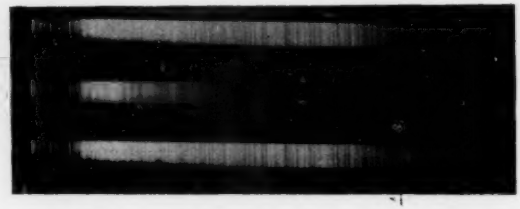

Fig. 7.

I wish to draw attention to the number of cells, 80 per c.mm., the increased protein and the gold curve, which I believe is called the meningitic curve and which in my experience is rather a rare type.

Fig. 8 is also from a case of tuberculous meningitis, and fig 9. as well.

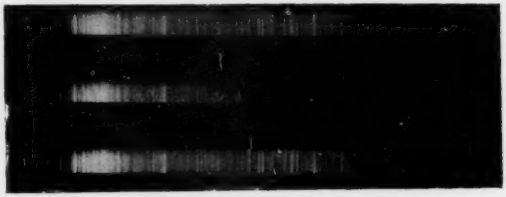

Fig. 8.

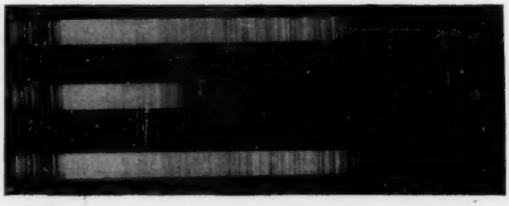

Fig. 9.

This type of spectrogram is very striking, and it is evident that such a large amount of absorption must depend on some change in the chemical or physical constituents of the fluid.

The obvious suggestion that increase in the cell count might account for it was investigated, but fig. 10, which is from a case of general paralysis in which the cell count was almost identical with the last and yet in which the absorption is quite different, shows that such a hypothesis is untenable. Nor can the absorption be wholly dependent on increase in the amount of protein since in both the fluids depicted the protein is equally increased.

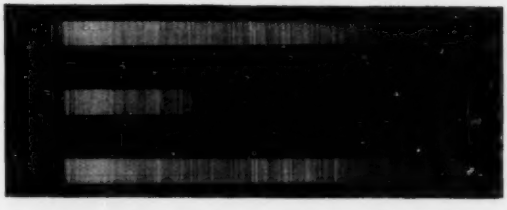

Fig. 10.

The Lange test in these two fluids gave very strikingly different results and the last case gave the most marked paretic curve I have seen. However, I will return to this point later. Meanwhile I have depicted a summary of these two cases, as they both represent ' type ' spectrograms and pathological findings in the fluid in the next figure (fig. 11).

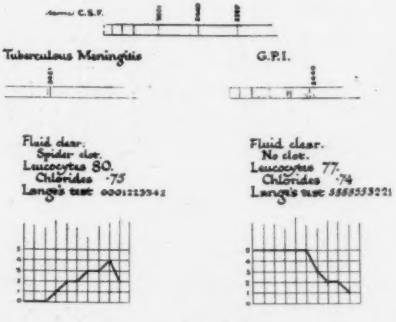

Fig. 11.

To return to the consideration of tuberculous fluids. I found that the spectrograms I obtained last year using a copper arc were corroborated in this second series of observations and I was beginning to feel that a spectrogram

might be of some diagnostic significance, at any rate in cases of suspected tuberculous meningitis. Then in November last (1929) I examined the fluid from a case which had been admitted as a suspected tuberculous meningitis and obtained a spectrogram which did not agree with my previous cases. This particular spectrogram is shown in fig. 12.

It will be seen that absorption begins about the same place as in the other tuberculous cases, though as a matter of fact it is really a little further along the band, the actual wave-length value being 2937 a.u. But instead of the usual absence of any further lines, in this spectrogram there are to be seen a number of lines further along actually in the region of 2702 a.u. to 2440 a.u. From this I suspected that the case was probably not one of tuberculous meningitis. The clinical history lent some colour to this suspicion, since after a very short illness, a few days only, the patient developed a bilateral choked disc, headache and vomiting, with some slight paresis of a left hemiplegic type. The pathological report on the spinal fluid was also not without ambiguity, for

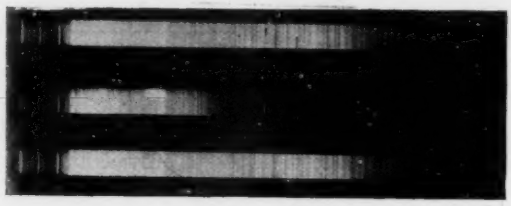

FIG. 12.

although the cells were increased to 520 per c.mm. with a 90 per cent. mononuclear count the chloride content was not diminished, which I have come to look upon as unusual in tuberculous meningitis.

After a few days' observation therefore, with an increase in the swelling of the optic discs, in view of the absence of 'diminished chloride' content, a suspicion of cerebral tumour was raised and as there was a definite though slight left-sided paresis, it was considered justifiable to carry out a right-sided temporal decompression. Although there was a slight improvement in the general condition for a day or so the patient quickly began to lose ground and died ten days after admission. At the autopsy the original diagnosis was proved to be correct, there being "some miliary tubercles scattered over the base of the brain and the vertex of the cerebellum."

There was no doubt as to the diagnosis and I was disconcerted at finding my previously conceived view of the definiteness of the spectrogram in this condition upset; but since this observation I have seen two other cases of tuberculous meningitis established by autopsy in which the spectrogram has been similar to the last, as is seen in the next two figures (figs. 13 and 14).

In fig. 13 absorption commences at 2937 and then a few lines are seen about 2764 to 2440, almost identical with the last plate.

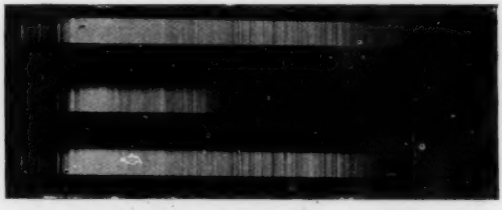

FIG. 13.

In fig. 14 absorption commences at 3051, the usual place for tuberculous meningitis, and then after a long interval a few lines are to be made out about 2702 a.u.

FIG. 14.

Although I was disconcerted at these readings there could be no shirking them; and though I did my best to make them fit in with my previous results the discrepancy was too obvious, so I had to accept them and realise that my ideas on the fixity of type of the spectrogram in tuberculous meningitis were wrong.

The next setback to these ideas occurred a few weeks after taking the last spectrogram, when I admitted an emergency case to the Nursery Ward with an outside diagnosis of purpura. Two days after admission it was clear that some meningitis was present and a lumbar puncture was done, giving exit to a turbid fluid on which the pathologist reported as follows:—

Turbid fluid.
Red cells 0.

Leucocytes 132,000	Polymorphs	85 per cent.
	Lymphocytes ..	13 per cent.
	Endothelial cells ..	2 per cent.

Protein much increased.
Globulin test positive.
Chlorides .. .. ·75 per cent.

Although no organisms were found in direct films a pure growth of meningococci was obtained and a diagnosis of cerebrospinal fever definitely established.

Fig. 15 shows the spectrogram obtained in this case.

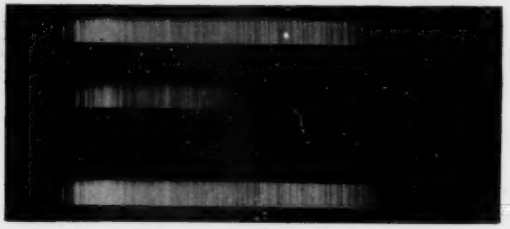

FIG. 15.

It will be seen that this is very similar to those of the last three cases of tuberculous meningitis. Absorption begins about the same place, viz., 2937, and then after a short interval there are further lines to be seen from 2702 to 2440 a.u.

I will complete this short series of cases by one more. It was that of a child of five admitted moribund one evening suffering from meningitis following otitis media. A cistern puncture was done as part of an attempt to flush out the subarachnoid spaces with saline and serum, and the spinal fluid obtained was reported on as follows :—

Turbid fluid, clotting spontaneously.
Leucocytes 2,180 per c.mm. Poly. .. 75 per cent.
Lymph. .. 25 per cent.
Protein much increased.
Globulin test positive.
Chlorides .. .. ·67 per cent.
Gold 0112222110
Hæmolytic streptococci isolated from fluid.

The child died 24 hours after admission.

Fig. 16 shows the spectrogram obtained in this case.

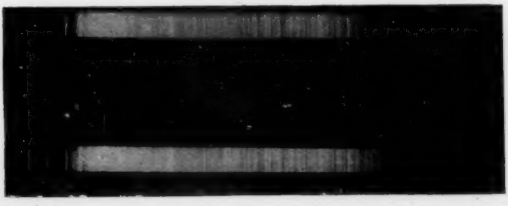

FIG. 16.

This fluid was so purulent that it is very difficult to make out where absorption really commences, but careful measurement shows it to be about 3951, i.e., the region at which absorption usually begins in tuberculous meningitis.

I thought it might be worth while to centrifuge this fluid which I did at high speed for ten minutes and on re-examining the centrifuged fluid I obtained the spectrogram shown in fig. 17, which will be seen to be almost identical with the atypical spectrograms from tuberculous meningitis.

On reconsidering this group of cases of meningitis I had to admit that my previous view of the spectrogram of tuberculous disease was too narrow and in trying to find some common factor I took into consideration the length of time elapsing between examination of the spinal fluid and death or other termination of the case, since my conclusion previously was that " the nearer a fatal termination the more complete is the absorption."

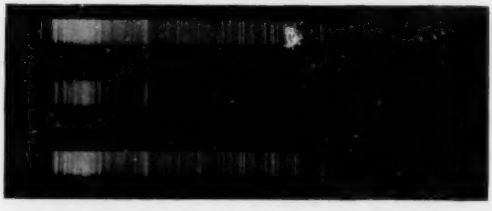

FIG. 17.

It appears just possible that there is some relationship between this interval and the type of spectrogram obtained, the details of this small series from this point of view being as follows :—

T.B. Cases absorbing beyond	3051	death within 3 days.
Strep. Case absorbing beyond	3051	death within 1 day.
T.B. Cases with partial absorption	2937	death within 10 days.
C.S. Fever with partial absorption	2937	non-fatal.

Thus the cases of delayed fatal tuberculous meningitis show a few lines in the spectrum a little further along, as does also the fluid from the non-fatal case of cerebrospinal fever and also as does the centrifuged fluid from the case of streptococcal meningitis. I am therefore tempted to suggest that in cases of meningitis there is a ' type ' spectrogram in which absorption begins at about 3051, is more or less complete for a short distance and then shows a few more lines about 2764 to 2440 ; if there be any tendency to improvement then this interval from 3051 to 2764 tends to fill up with lines, whereas if, as is usual, the case progresses to a fatal termination not only does this interval never show any line but those about 2764 disappear, so that total absorption occurs from 3051.

It might thus appear that the spectrogram has some prognostic value according as the interval between 3051 and 2764 tends to widen or fill up in a series of fluid examinations. Fig. 18 shows this interval actually in the process of so filling up. This spectrogram is from the case of cerebrospinal fever 14 days after the first one was taken.

FIG. 18.

CONCLUSIONS.

To conclude, first of all I feel that formerly I stressed too much the individuality of the spectrogram in any of the conditions I examined; and, secondly, I would modify my previous opinion by saying that in cases of meningitis of any type there appears to be a certain selective absorption with an interval between wave-lengths of 3051 and 2764 and that variation from this type depends on the natural history of the individual case.

I have tried to depict this view in fig. 19 in which the middle band represents the 'type' spectrogram whilst the upper and lower represent the variants of it.

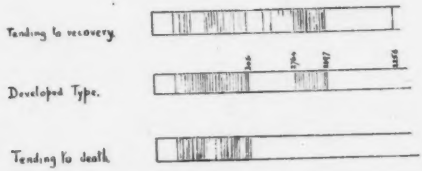

FIG. 19.—Diagrammatic representation of spectrograms in meningitis.

Whether the above conclusions are warranted or not will remain for more extended observations but I am inclined to offer a speculation that the absorption depends on some physical factor in the fluid which is at present undetermined.

When one looks at the perfectly clear fluid from a late case of tuberculous meningitis and an equally clear fluid from a case of tabes or general paralysis and in both finds a pleocytosis, an increased protein content, and an identical salt content and yet with spectrograms very different in their appearance,

the conclusion seems fairly legitimate—either our means of chemical investigation are too gross to detect the minute differences in composition on which the spectrograms are dependent or, what is more likely, the differences depend on factors other than chemical.

If the reader will refer to the two first figures and their epitome in fig. 11, he will see the only difference in their pathological reports lay in the Lange gold test.

Lange's test almost certainly depends on an altered electrical state in colloid particles in the mixture of spinal fluid and gold chloride, and the so-called 'curves' obtained in various clinical conditions may not be much more specific than the spectrograms I have shown. I think it not unlikely that both phenomena depend on similar though, of course, not identical variations in the surface charges of particles of colloid.

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STUDIES IN SPEECH NEUROLOGY—I.*

By

Professor E. W. SCRIPTURE, University of Vienna.

INTRODUCTION.

Speech neurology may be defined as the study of the phenomena of the nervous system underlying the production of speech. It is based on the registration of speech in such a manner that its factors can be accurately measured and analysed.

The first registrations of speech for the purposes of neurology were made by the author in the Psychological Institute of the University of Münich in 1905 at the suggestion of the Director, Professor E. Kraepelin. From 1906 to 1912 the work was carried on in the neurological department of the Vanderbilt Clinic in New York City. The work was then removed to London where it was carried on for several years in connection with the Out-Patient Clinic of the late Dr. F. E. Batten at the National Hospital. From 1923 to 1928 it was pursued in the Psychiatric Institute of Professor Wagner-Jauregg in connection with the Phonetic Institute of the University of Vienna. From the summer of 1928 onwards it has been continued in connection with the Out-Patient Clinic of Dr. Kinnier Wilson at the National Hospital, London. At the present time similar investigations are being made in the completely equipped speech laboratory of the West End Hospital for Nervous Diseases, London (Director, Dr. C. Worster-Drought; Assistant, F. Janvrin) and in the Experimentalphonetisches Laboratorium der thüringischen Landesheilanstalten, Stadtroda (Director, Prof. W. Jacobi).

Speech neurology had its origin in the application of the methods of registration developed by Rousselot for the purpose of studying languages and dialects. His work led to the development of the science of experimental phonetics; this has now been extended to most branches of the study of speech. In order to afford opportunities for united action of the workers in this science the *International Society of Experimental Phonetics* was founded at The Hague in 1928. A special section for the division of speech neurology was created in 1929 with Dr. Kinnier Wilson as Chairman. Members of this Society receive free of charge: *Zeitschrift für Experimentalphonetik, Vox, Comptes rendus de la Societe Internationale de Phonetique Expérimentale, Bulletin of the International Society of Experimental Phonetics* and *Sprachneurologische Mitteilungen*. Applications for membership are to be sent with the yearly fee of 10s. to the *Secretarial Bureau*, 73 Welbeck Street London, W.1. A meeting of the Section is being arranged in connection with the International Congress of Neurology at Berne in 1931.

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MAKING AND INTERPRETING SPEECH INSCRIPTIONS.

The patient speaks into a wide mouthpiece with a rubber air-cushion (fig. 1). The vibrations and movements of the air pass down a wide rubber tube to a membrane of oil-silk in a speech recorder. The movements of this membrane are enlarged by a straw lever and inscribed on a surface of smoked paper stretched around a revolving cylinder. Before or after a series of records a time-line is registered by a fork vibrating 100 times per second. The record sheet is finally removed and fixed.

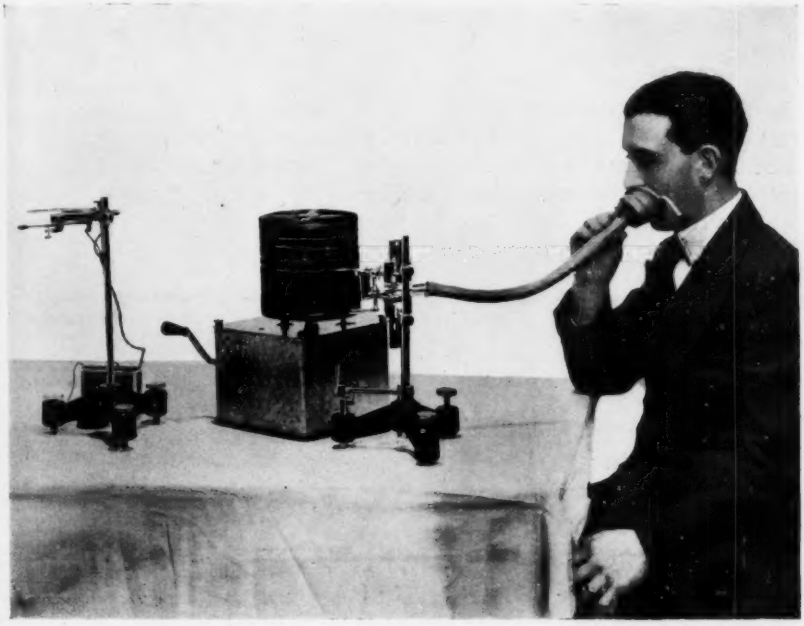

FIG. 1.

A speech inscription of ah spoken rather long by a normal person is reproduced in fig. 2 (E.S.). The height of the line of waves depends on the expenditure of breath. Each wave is the registration of one puff from the larynx. The vertical height of the waves gives some indication of the strength of the puffs. The horizontal length of a wave registers the time from one puff to the next.

Under a microscope the horizontal length of each wave is measured. The results are turned into frequencies, that is, the times with which each vibration could be repeated in one second. Horizontally long vibrations correspond to low tones, horizontally short ones to high tones. The frequencies are then

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FIG. 2.

plotted on millimetre or logarithmic-millimetre paper. A line drawn through the dots gives the course of frequency, or the melody plot. The melody plot for fig. 2 (E.S.) is given in fig. 3 (E.S.). The tone on the whole neither rises nor falls but remains at about the same pitch. It is particularly important to notice that the tone never remains exactly on the same pitch but fluctuates continually.

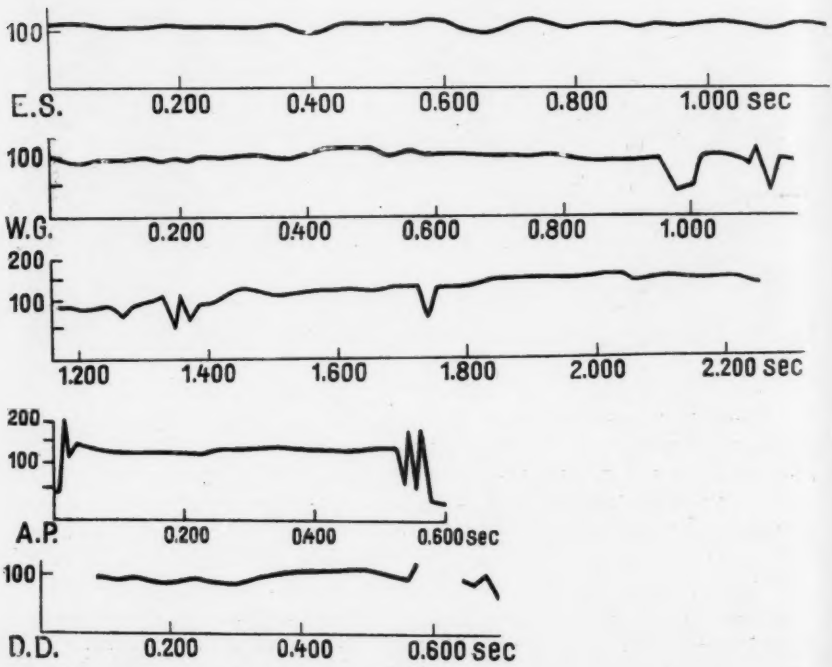

FIG 3.

EXAMPLES OF SPEECH ATAXIA.

All the patients referred to in the following account were from the Out-Patient Clinic of Dr. Kinnier Wilson at the National Hospital in London.

CASE 1. The inscription in fig. 2 (W.G.) is from a case of *cerebellar agenesis*.

> W. G., 24 years old, male, had been affected with shaking of the head and general tremor since childhood. His father, a sister and one brother were similarly afflicted. He holds his head tilted to the right; it shakes intermittently. His hands shake, but he shaves himself and does not spill tea. His tongue does not seem tremulous. There is no disturbance of the eyes. The Wassermann test is negative in the serum. A circle drawn by the patient with a pencil is reproduced in fig. 4.

FIG. 4.

In the inscription in fig. 2 (W.G.) the height of the wave line above the base registers the breath expenditure. In general it is scarcely raised; this indicates a slight expenditure. At one point it rises and falls suddenly; this indicates a momentary increase of breath pressure. The course of the breath expenditure is therefore irregular. Such an irregular result of muscular movements is said to be *hypotaxic* or *ataxic*.

The vertical height of a wave registers to a great extent the amplitude or strength of the puffs from the larynx. In general the waves of the inscription in fig. 2 (W.G.) are of fairly constant height. At the place just considered they suddenly become higher. This is evidently another expression of the disturbed regulation, that is, of hypotaxia.

The horizontal length of a wave in the inscription is a registration of the time between puffs from the vocal lips. The inscription is characterised by the frequent occurrence of great irregularities. This is still another expression of the hypotaxia.

The melody plot in fig. 3 (W.G.) in its general course is a characteristic one for ah spoken at great length or sung. The frequency remains fairly constant for long regions at a time; the gradual rise towards the end is not abnormal. The small smooth fluctuations in frequency are always present in normal cases. The fluctuations in breath pressure are the same as in a normal wavery or vibrato voice. The sudden jerks in the plot are due to the irregular waves and are a record of the hypotaxia. The melody shows no abnormality except the hypotaxia.

The amount of the breath expenditure and the loudness of the vowel depend on the action of the breath muscles; the pitch of the vowel waves depends on the tensors of the larynx. These activities depend on the co-

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ordination, that is, on the *synergia*, of the agonist and antagonist muscles. The irregularities in the speech, the hypotaxia, arise from defective co-ordination or *hyposynergia*. This is expressed by the kineto-dynamic causal equation

hypotaxia ←—hyposynergia.

This is the motor equation for cerebellar agenesis.

CASE 2. *Disseminated sclerosis.*

A. P., male, 46 years old, complains of losing the use of his legs; they always feel cold, the left leg feels numb; the hands feel cold and he has poor use of them; knee and ankle jerks are increased, the plantars are extensor, the abdominals are absent; there is a very slight nystagmus, the finger-nose test is positive. No optic disc changes. No defect of speech can be heard; the voice seems to have a somewhat melancholy melody, slowing rising or falling. Diagnosis: *disseminated sclerosis* with spastic paraplegia as the most striking manifestation.

An inscription of ah spoken by this patient is reproduced in fig. 2 (A.P.). Long irregular waves appear at the beginning and at the end, that is, when the voice is beginning or ending a tone. The resemblance to the results for the usual finger-nose test suggest that this may be termed an intention tremor of the larynx. The melody plot in fig. 3 (A.P.) shows a rather monotonous melody with sudden changes due to the irregular waves. The condition is one of *hypotaxia* and *hypomelodia*. No other abnormalities are found in the inscription. The hypotaxic waves are due to laryngeal hyposynergia and the hypomelodia to overtension of the laryngeal muscles. The motor equation for the condition is therefore

hypotaxia + hypomelodia ←—hyposynergia + hypertonia.

CASE 3. *Friedreich's Ataxia.*

D. D., male, 27 years old, had for six years noticed uncertainty in walking, weakness of arms, and slowness of speech; only lately has he had hesitation in micturition. Disturbance of vision has not been noticed. He is slightly melancholy; a sister is said to be somewhat mentally abnormal; otherwise a normal family history. The muscular power is good. There is marked kyphoscoliosis and also talipes equinus with pes cavus on both sides. The pupils contract to light but widen again. There is no nystagmus; fundi normal. The vibration sense is lacking in legs and arms. The finger-nose and heel-knee tests show ataxia. Adiadocokinesis present. Knee and heel reflexes absent. Plantar reflexes extensor, both sides. Diagnosis: *Friedreich's ataxia.*

An inscription of ah spoken by this patient is reproduced in fig. 2 (D.D.). The height of the general line shows wavering of the breath pressure at the beginning and the end. Strongly irregular waves appear at the end. There is thus *hypotaxia* in both breath pressure and voice tone. The melody plot is given in fig. 3 (D.D.). The irregularities at the end indicate hypotaxia. The general course of the tone shows deficient modulation and flexibility. There is therefore no hypomelodia. The motor equation is:

hypotaxia ←—hyposynergia.

In none of the cases could any abnormality of speech be detected by the ear. The test was therefore ultra-acoustic. Its invariable presence in disseminated sclerosis although undetected except by these methods suggests the

hypothesis that in this disease every portion of the nervous system may be affected from the start, and that when nystagmus, intention tremor, etc. are apparently not present, this is simply because the methods of observation are not fine enough to detect them.

EXAMPLE OF SPEECH ASTHENIA.

K. N., female, 49 years old, noticed two years ago that her hands went blue in winter. One year ago she noticed that the upper lip could hardly be moved in eating and speaking; somewhat later the jaw would fall down during sleep. She now complains of weakness in the arms and legs and tiredness in the eyes when reading. At night her eyes take a crossed position and she must move her head to and fro in order to get them into a comfortable position. Double vision occurs occasionally. The eyelids fall when she is tired. In looking sidewise there is a feeling of strain. In the theatre she has to look down often for rest. The ocular movements are not defective. Nystagmus is not present. In attempting to smile the upper lip remains immovable. The tongue is protruded properly; it shows the three typical myasthenic longitudinal grooves. There is some difficulty in swallowing; fluids return through the nose. All the muscles of the body are hypotonic but not wasted. The palate is scarcely movable. All reflexes are normal. The speech appears indistinct and nasal. Diagnosis: *myasthenia gravis.*

Requested to repeat *pa—pa—pa* continuously without interruption, the patient furnishes speech inscriptions of the kind shown in fig. 5. The

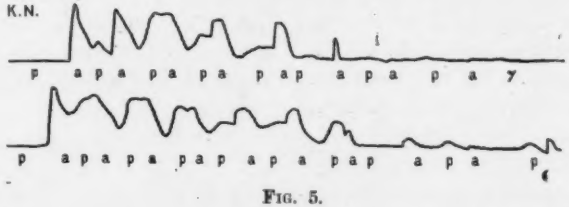

FIG. 5.

inscription begins with a straight portion for the stoppage during the closure of the lips for *p* followed by a sharply rising line for the explosion at the opening of the lips for *p*. The descending line with small waves that follows is the registration of the vowel. The second registration of *pa* shows a defective closure and a somewhat weaker explosion. The third *pa* shows a blast of air (from open lips) instead of a closure; it is, in fact, a puff of air between two vowels. The records steadily become weaker and more defective until they die away in an almost formless murmur. After a brief pause another series of syllables starts strongly, only to become defective and fade away likewise. This is repeated as long as the patient attempts to speak.

GENERAL CONSIDERATIONS.

The immediate value of speech inscriptions lies in their use for diagnosis. The making of the inscription can be made by a trained laboratory assistant; the presence of the physician is not needed. When the typical inscriptions for various diseases have been definitely established, the diagnosis can be made by

comparison with a chart of results. This is true, for example, with voice ataxia. The peculiar waves that are found in all cases are never present in any other condition. An inscription with such waves automatically gives a diagnosis of laryngeal hyposynergia, leaving to the physician the further decision whether the case is one of disseminated sclerosis, cerebellar lesion, Friedreich's disease, etc. Great value arises from two facts: (1) that the diagnosis is infallible, and (2) that it can be made when no alteration in the voice can be detected by the ear. Of interest is the fact that the presence of such waves in an inscription often prevents an erroneous diagnosis.

The greatest value of speech inscriptions lies in their application to the study of the mechanism of the nervous system. Only a few suggestions can be made here. The first step is to introduce accurate notions and terminology in place of the present confusion. In the preceding account the term 'taxia' has been applied to the regular condition of the movement resulting from the activity and the term 'synergia' to the muscular action itself. Hypotaxic inscriptions result from hyposynergic muscular activity. It is certain that hypotaxic inscriptions may result from hyposynergia arising from lesions at various levels. It is not yet possible to make any final statements; it can only be said that the inscriptions already obtained indicate the possibility of disturbances of synergia in the centres on the floor of the fourth ventricle, in the cerebellum and perhaps in various nuclei. A further step is the investigation of the nervous mechanism itself. The statement that hyposynergia is due to disturbed action of the nerve-cells is not enough. It may be possible to determine the nature of this interaction.

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For apparatus: F. Homan, 13 Florence Road, London, S.E.13.

Editorial.

THE NEUROSES.

NOTWITHSTANDING advances that are taking place all along the front of medicine, it is a question whether present-day conceptions of what are currently denominated 'neuroses' and 'psychoneuroses' are as serviceable as is often claimed. To classify and differentiate the neuroses has ever been troublesome, mainly though not entirely owing to etiological uncertainty. Old distinctions between 'functional' and 'organic' types of symptom have proved valueless except for clinical diagnosis, and in this field also their usefulness is over. If the Babinski variety of plantar reflex response characterises a series of conditions whose basis is an actual structural change in cerebrospinal tracts, it is also found, unmistakably, under circumstances which no one can say represent other than transient disorders of neural function. For a few minutes after a major epileptic attack the plantar response may assume the form of typical great-toe extension. From the standpoint of causation, therefore, a test so well-recognised and constantly employed as that of plantar stimulation cannot render any aid.

It may become difficult, accordingly, to find objective criteria whereby to define and determine a 'neurosis.' Theoretically and etymologically, the term, as Dr. Millais Culpin urges, should indicate a disturbance of the intrinsic function of nerve tissue—it is pointless otherwise. But what is this intrinsic function, we may well enquire? Excitation and inhibition are the physiological phenomena of neural tissue; stimuli applied to the nervous system elicit responses which assume the form of the one or the other, or of their succession, nor can any other variety be obtained. Thus considered, any neurological syndrome is a neurosis. The vicissitudes through which the word has passed, however, impress us to-day with the desirability of a certain restriction; we find, in point of fact, that it is commonly utilised to signify disorders of function for which, by way of cause, no recognisable physiological disability can be detected. In the seeming absence of such etiology, present-day doctrine

declares for a psychological origin, and some, e.g., Dr. Culpin, go so far as to consider neurosis synonymous with 'minor psychosis.' Others advance further; Dr. Israel Wechsler includes the psychoses (paraphrenia, schizophrenia, manic-depressive psychosis, and paranoia) under the term 'narcissistic neuroses.'

Here, surely, is confusion confounded. And yet the steps whereby so unsatisfying a nomenclature is reached can be readily followed. For the truth is that 'neurosis' is merely a name, an attempt to describe (for the sake of the clinician) a series of states which it is thought must be called something and might as well have that label attached as any other. In the consideration of clinical conditions for which the designation seems appropriate two matters have to be examined—firstly, the causation, and secondly, the intrinsic nature of the symptoms. Now as regards the former of these, it behoves the critical mind to proceed warily. The terminologists of the day appear with some unanimity to accept say writers' cramp as an 'occupation neurosis' and to argue for its inclusion because 'examination and experience tell us that its subject has no physiological disability of nerve.' It is alleged to be analogous to stammering —and that is of psychogenic derivation, we are assured. But so far as the manifestations of writers' cramp are concerned, they are in fact capable of being described as expressive of either increased excitability and irritability, on the one hand, or of augmented inhibition on the other. That is to say, their intrinsic features indicate disorder of physiological function, postulated psychical origin notwithstanding. Thus a case might be made out for the view that this particular 'neurosis' may develop on a basis of inherent defect of neural function, showing itself only when a given set of circumstances (e.g., long repetition of the same muscular act) is in being. Or take 'la grande névrose' of our French colleagues, the hysteria whose fundamental problems, *pace* the psychoanalysts, have not yet been solved. If a patient cannot move his arm, which hangs flaccid from the shoulder, and if examination discloses no change in electrical reactions or reflectivity, are we to say that its physiological function is normal and that the condition constitutes a neurosis, a 'minor psychosis'? On the contrary, from the viewpoint of physiology it is one of pure inhibition. The person concerned cannot innervate his brachial neuromuscular units; there is a block somewhere. What boots it to ascribe dogmatically the impasse to the psyche? This is surely an assumption of an unwarranted kind. For we do not

know what the psyche stands for in physiological terms, and if the psychologist smiles at the naivété of imagining the two systems comparable it is none the less conceivable (evidence pointing in this direction might be adduced) that the ' will ' is nothing else than de-inhibition, and that the failure of the hysteric to innervate his arm is a question of physiological incapacity to remove inhibitory impediment. That this neural defect—for such it is—should be conditioned by a disorder of the psychical series may be the case ; the symptom arises, at any rate, on a mechanistic basis capable of physiological description. Nor must we too rashly conclude that were a psychical trauma or other such impairment to be discovered in the patient's mental history the exteriorisation of the syndrome is thereby explained.

No less deserving of reconsideration is the term ' psychoneurosis,' to find a proper justification for which would be a matter of no little arduousness. Like ' psychomotor,' ' psychosensory,' ' psychovisual,' and others, it represents a blend of physiological and psychological without any definition of the assumed interaction or interrelation. If it stands for something a little more than neurosis, a little less than psychosis, its descriptive value is outweighed by its nosological impropriety. For, as we have seen, one of the disciples of present-day modernism (the collocation is intentional and meaning-full) takes neurosis to signify psychosis—' minor,' if you will, but who is to define ' minority ' ?

Until some agreement is attained in respect of the principles on which our terminology in medicine is to be established confusion in the names given to things will not be dispelled ; further, persistence in employment of inappropriate designations will continue to imply the existence of unfounded etiological conceptions. The word ' neurosis ' implies disorder of neural function—it is therefore essentially unspecific ; if it entails the admission of a psychical causation it is unsuitable ; and so long as it is imagined to stand for physiological disorder with physiological integrity it is meaningless. Of more immediate practical importance than all such discussions is the problem whether any particular physiological characters distinguish the neurosis from other functional imperfections. This aspect of the matter, still largely unnoticed, demands painstaking research. Even those who contend stoutly for the psychological origin of the neuroses (as they understand the term) must naturally admit the frequency with which actual interference with physiological mechanisms occurs. The ' anxiety neurosis ' of current textbooks is compact of recognisable and definable somatic and visceral disturbances.

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In themselves, have these such a stamp, do they run such a course, or exhibit such intrinsic features, as to enable the physician to separate them from other involvements of the same mechanisms ? Can any peculiarities be discovered in its physiological manifestations to differentiate hysteria from other states ? The limited possibilities of the psychical-sexual origin of hysteria must contrast strikingly, in the mind of any unbiased observer, with the remarkable variability of its manifold clinical phenomena. Were any specificity to attach to these, from a neural point of view, the inadequateness of psychogenic theory would be patent. The more deeply clinical observation penetrates the arcana of the neuroses the more possible does it seem that the class as a whole will suffer dismemberment. Chorea was once a neurosis, and paralysis agitans ! Have we finished with telegraphists' cramp when we note its occurrence in a 'neurotic' person, with 'nervous heart,' bad dreams, fatigability, fears and what not ? What if these are themselves but the expression of physiological disarray ?

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[45] **Superior olivary nucleus.**—J. W. Papez. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 19.

Lesions were made through the base of the skull in and around the superior olives in a number of cats to determine the fibre connections of these organs. After eleven days, the brains were treated by the Marchi method for fibre degenerations and were sectioned. From a study of these sections, the following conclusions are made :—

1. The acoustic striæ cross through the vestibular nuclei, enter the reticular formation and divide into two fibre streams, one that enters the lateral side of the olivary peduncle of the same side and joins the lateral S-shaped segment, and the other that crosses the raphe forming the decussation of the striæ and joins the S-shaped segment of the other side. Both streams continue upward to the inferior colliculi.

2. A lesion of the medial segment of the superior olive or of the adjoining medial preolivary nucleus or the nucleus of the trapezoid body causes a degeneration of the medial fibres of the olivary peduncle passing dorsally through the abducens nerve across the midline and out on the other side by crossing over the descending root of the trigeminus. They appear to join the vestibular nerve.

3. From these experiments it also appears that no longitudinal fibres are contributed by the superior olive into the reticular formation dorsal to the superior olive such as have been postulated by Cajal. In other words the olive does not appear to play the role of a reflex centre.

4. All fibres arising from the superior olive, except the peduncle, appear to be contributed to the lateral lemniscus.

5. The superior olives do not appear to be connected with the cerebellum as was thought by Bechterew and others.

6. An injury of the ventral reticular nucleus of the medulla oblongata and pons produces a constant degeneration of the reticulocerebellar fibres of van Gehuchten, which enter the floccular lobes by passing ventral to the dorsally turning limb of the restiform bodies.

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7. An injury to the roots of the abducens nerve produces a loss of the nictitating membrane reflex or bulbar retraction. Likewise, a lesion in the trigeminus nucleus lateral to the outgoing limb of the facial causes a loss of the same reflex. R. M. S.

[46] **Extensor tonus after transections of the brainstem at varying levels.**—S. W. Ranson and J. C. Hinsey. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 584.

Decerebrate rigidity is more marked after transection of the brainstem at the anterior border of the pons than when the section is made through the caudal border of the mamillary body. It is usually even more marked after anæmic decerebration, by which all of the brain anterior to the pons is rendered functionless. Cats which after section of the brainstem on a plane extending from the anterior border of the superior colliculus to the optic chiasma are able to walk, show extensor rigidity when supported with the legs pendent, although this is often masked by running movements. When resting in a crouching position these cats show some increase of tonus in both flexors and extensors. Complete removal of the red nucleus is compatible with the development of the highest grade of decerebrate rigidity. This nucleus is therefore not the source of the impulses responsible for this rigidity. Its influence is exerted rather in the direction of inhibition of extensor tonus, as has been shown by Rademaker. But it is not the only centre concerned in this inhibition, as is demonstrated by the fact that exaggerated extensor tonus develops in hypothalamic cats when these are supported with the legs pendent. Under these conditions the rigidity appears to be due in part at least to the cutting off of impulses from the cerebral cortex. Plasticity is present in transections either above or below the red nucleus and after anæmic decerebrations, but is most easily demonstrated after high transections. R. G. G.

[47] **The problem of localisation in experimentally induced convulsions.**—F. H. Pike, C. A. Elsberg, W. S. McCulloch and M. N. Chappell. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 847.

The question of the site of origin of the clonic and tonic elements of convulsive seizures has been much debated. Much experimental and clinical evidence has been adduced to support the contention that the cortex is the source of the clonic and that subcortical mechanisms are responsible for the tonic parts of the motor disturbances, but it has been demonstrated experimentally that clonic convulsions can be produced in animals after the total excision of the cortical motor areas of both sides of the brain. The more closely the question is studied, the clearer it becomes that the problem of the convulsive movements is only a special case of the more general problem of the functional organisation of the motor system as a whole.

From their experiments on animals the authors believe that, normally, the pyramidal is the main motor system which is responsible for the clonic convulsions that occur when the pyramidal system is entirely or almost entirely intact. Tonic convulsions arise from the lower motor mechanisms in the period immediately succeeding an injury to the cortical motor mechanisms. After the lapse of sufficient time, clonic responses may be elicited from them. This is, however, not evidence that clonic convulsions may and do arise from the lower motor mechanisms when the cortical mechanisms are intact.

Tonic convulsions are absent in the early postoperative stages in animals in which the midbrain has been split longitudinally in the median line, while clonic convulsions persist if the pyramidal system is intact. All parts of the motor mechanism act together as one system when the brain is intact. It does not seem probable that when the whole motor system is intact, one part of the mechanism gives rise to movements of one type and some other part of the mechanism independently gives rise to movements of another type.

R. M. S.

[48] **Sleep as a problem of localization.**—C. v. Economo. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 249.

The author discusses the evidence both pathological and physiological in favour of a centre for the regulation of sleep. On the whole he thinks himself warranted in postulating a point at the junction of the thalamus and interbrain which though not in itself a localized centre regulating sleep is the nodal point of an area of grey matter concerned with this function. If such be the case he considers it of importance not only as an aid in the localization of disease more especially of tumours, but also because he thinks it may be possible to influence such a centre by radiation or diathermy, and so treat insomnia by methods which he regards as superior to drugs, physiotherapy or psychotherapy.

R. G. G.

NEUROPATHOLOGY.

[49] **Normal and pathological structure of microglia** (Recherches sur la structure normale et pathologique de la microglia).—G. Marinesco. *Ann. d'anat. pathol.*, 1930, vii, 153.

This article gives a fairly full resumé of the work on the microglia which has been done in Spain, America and Germany, and adds several facts which have been brought to light by Roumanian studies under the author's direction. The normal distribution and arrangement of the microglia in various parts of the central nervous system of man and the common experimental animals is described more completely than is usual except in the original Spanish articles of Hortega. In addition a short note is given on the microglia of some cold-blooded animals.

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In this connection it is worthy of note that the developing microglial cell passes through a stage in which it contains fatty granules similar to that found by Wells and Carmichael in tissue cultures of analogous cells from the fowl. A full description is also given of the changes in the microglia in a great variety of diseases, among which we note ischæmia, rabies, herpetic and lethargic encephalitis, borna, vaccinia, septic meningitis, disseminated sclerosis, and neuroptico-myelitis. In general paralysis, in addition to the well-known formation of rod-cells, various other changes are described including the formation of giant-cells, and of rosettes which sometimes encircle a giant-cell. Changes are also seen in the microglia in poisoning by arsenic, lead, morphine, veronal and coal gas. These are rather indicated than described, as are also the alterations in such chronic degenerative diseases as senile dementia, dementia præcox, Huntington's chorea, and epilepsy. The author has also studied the microglia in a juvenile case of amaurotic family idiocy, in which he found many stages towards the formation of fat granule cells. Mention is also made of the author's recent studies of the microglia in senile plaques and in experimental replacement of the cerebrospinal fluid by Ringer's solution of varying hydrogen-ion concentration.

The article is thus more a review of recent literature than a fresh contribution to the subject, but the fact that much of the author's own previous work and that of his collaborators has been published in Roumanian, as well as the wide scope of the present article and the numerous references given, make it a useful contribution to our knowledge of the microglia.

J. G. G.

[50] **Niemann-Pick's disease.**—G. B. Hassin. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 69.

The histological changes recorded exhibited great similarity to those seen in the various types of amaurotic family idiocy. There were differences, of which the most striking were the changes in the cerebellum. Marked as the latter are in some late infantile types of amaurotic family idiocy, they were much more marked in the case recorded. The prevalence of glia tissue and blood-vessels in the various cortical layers, the extreme scarcity of Purkinje cells, the density of the fibrous glia and the abundance of neutral fat substances in the perivascular spaces of the blood vessels suggest a far advanced parenchymatous lesion with the transformation of the cerebellar cortex and optic thalamus into the so-called glial scar. One gains the impression that in this disease the pathological phenomena are much farther advanced than in ordinary amaurotic family idiocy, though the fundamental type of changes is generally the same. Here one has the same expansion of the ganglion-cell bodies with practical obliteration of the dendrites, the identical accumulation in them of prelipoids, and the same pronounced cerebellar and thalamic changes. The microchemical changes also are similar and denote that the conditions under discussion are

members of one pathological group. As Bloom well puts it, Niemann-Pick's disease appears to be "a profound disturbance of metabolism in which the infants do not thrive; there is a piling up of lipoid material, especially phosphatids, in phagocytic cells throughout the body while neutral fats are more or less gone or destroyed. In this they differ from Gaucher disease where no deep metabolic disorder is present, for such patients may live up to 59 years."

In other words, the two morbid conditions are due to a metabolic disturbance. However, one is so far hardly justified in considering Niemann-Pick's disease and amaurotic family idiocy one disease process. As Sachs justly pointed out, it is rather singular that no striking visceral changes such as are seen in Niemann-Pick's disease have been described in amaurotic family idiocy. This would be the case if they were one and the same condition. Nor do all cases of Niemann-Pick's disease exhibit cerebral changes typical of amaurotic family idiocy. However, the pathological material has not been sufficiently abundant, nor were the visceral organs in amaurotic family idiocy thoroughly studied. Until such studies are forthcoming it may be considered that the two conditions are caused by a metabolic disorder which in some instances affects preferably the central nervous system (amaurotic idiocy), in others preferably the visceral organs (Niemann-Pick's disease) and in the minority of cases, the two. R. M. S.

[51] **Intracerebral calculi.**—R. C. Buckley. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 1203.

While intracerebral areas of calcification are known to occur in many tumours, infections and abnormalities of the brain, there are few verified examples of the formation of actual calculi or 'brain stones.' In the author's case the patient had experienced for 23 years infrequent epileptic attacks, each being preceded by muscular twitchings in the lower part of the left leg.

Roentgenograms showed two dense calcified areas 1·8 and 1 cm. respectively in diameter lying deep in the right cerebral hemisphere in the vicinity of the ventricles. They remained unchanged in size, position and appearance during the twelve-year period of observation.

Death occurred suddenly from a large hæmorrhage in the left cerebellar hemisphere. Both calculi were found in the white matter of the right cerebral hemisphere near the cingulum. The larger stone was embedded in a smooth shining cavity the wall of which was made up of a dense mass of collagen fibres with an outer zone of fibrous astrocytes; the small stone was sharply marked off from the brain tissue by a dense connective-tissue wall.

As the patient showed postmortem evidence of other healed vascular accidents as well as an acute, fatal cerebellar hæmorrhage, it is likely that the calculi represented the end-result of an area of degeneration or hæmorrhage with consequent absorption of the necrotic material and calcium deposition. R. M. S.

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[52] **The anatomical substratum of the convulsive state.**—W. SPIELMEYER. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 869.

PATHOLOGICAL anatomy cannot give support to-day to the view that there is a 'genuine' epilepsy, but it does permit of conclusions being drawn as to certain functional phenomena which immediately precede the epileptic seizures, and from his study of the pathological changes which lead to the well-known sclerosis of Ammon's horn Spielmeyer concludes that vasomotor disturbances are effective in the mechanism of the epileptic attack.

In the earliest stages the nerve-cells of this region are ischæmic and surrounded by Hortega cells, while in the cerebellum very similar changes are present. It is important to note that the histological quality of the changes is not dependent on the etiological process itself, for that process does not affect the characteristic location of the lesion in Ammon's horn. On the contrary, it is the same histological picture that one finds in all the different symptomatic epilepsies with their different anatomical substrata.

Since exactly the same changes may be found in cases of cerebral arteriosclerosis, thrombosis and embolism it is obvious that organic occlusion of the circulation produces changes like those seen in epilepsy, and from this the author concludes that in epilepsy, also, an impediment to the circulation must have been present; this hindrance is first felt by those parts poorly supplied with blood, viz., Ammon's horn and the cerebellum, whereas in other parts of the brain with a better blood supply, a compensatory reaction to the disturbance is possible. R. M. S.

[53] **Functional circulatory disturbances and organic obstruction of the cerebral blood-vessels.**—F. HILLER and R. R. GRINKER. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 634.

THERE is a noteworthy tendency in present-day medicine to explain certain organic lesions on the basis of functional circulatory disturbances. In neuropathology, especially, the pathogenesis of many lesions that could not be ascribed to organic causes is now regarded as the result of functional disorders of the blood supply to the central nervous system. G. Richer has shown that slight stimulation of the vasomotor nerves results in a dilatation of the blood-vessels with acceleration of the blood stream. Stronger stimuli lead to vasoconstriction followed by dilatation of the capillaries, and when accompanied by a slowing of the circulation, prestasis with petechiæ usually occurs. This state frequently develops into a stasis. If such a stasis develops rapidly, agglutination of the blood-cells may occur. A stasis is reversible and is not accompanied by transudation of serum, whereas prestasis with retardation of the blood flow, corresponding to what happens in inflammation, is associated with transudation. With exudation of serum, diapadesis of erythryocytes may also take place, and this often characterises prestasis in the environment of a necrosis,

itself caused by complete stasis. Such a combination of the necrotic effect of stasis, as in a softening, with the resulting prestasis of the surrounding tissue, leads to a red infarction. When prestasis is not followed by stasis there results an incomplete necrosis with hyperplasia of the mesodermal and glial elements in the nervous tissue.

Applying these conceptions to the pathology of the nervous system Hiller, Grinker and Meyer have shown that the characteristic areas of softening in the brain caused by carbon monoxide poisoning occur on the basis of prestasis and stasis. In other words, functional disturbances of the blood-supply lead to lesions of the nervous tissue of a severity as great as one has been accustomed to attribute only to organic circulatory disorders, as in thrombosis and embolism. This conception has been of considerable value for the explanation of lesions of the brain in various forms of eclampsia, such as occur in pregnancy and certain cases of pertussis, but whether the circulatory disturbance in pertussis eclampsia is organic, i.e., due to air emboli, or functional can be decided only by studying a case in which the convulsions have been separated a sufficient time from the period of spasmodic coughing to preclude the possibility of air emboli. The authors have been fortunate to obtain such a case and for comparison describe another case which demonstrates the effect on the brain tissue of an embolic occlusion of a main branch of the middle cerebral artery.

In the case of pertussis eclampsia no evidence of meningitis or hæmorrhage was found and the authors believe that clinically and anatomically these two possible causes of convulsions probably seldom, if ever, occur. The convulsions and the underlying cortical damage took place long enough after the cessation of coughing to preclude the possibility of an air embolism, and although no thrombosis or other organic obstruction could be found the essential pathology corresponded to the softenings typically found associated with vascular occlusions. The type of lesion varied from ganglion-cell 'Erbleichung' to incomplete and complete softening, which points to a slowly increasing diminution of the blood supply. The lesions were confined to the cortical layers and involved them in foci. The cornu Ammonis was similarly involved, but the white matter was entirely spared. This electivity is explainable by the fact that these areas have a functionally different blood-supply from the white matter and deeper structures, and are notably more vulnerable to disturbances of their nutrition. Within the softened areas the cortical blood-vessels showed tremendous dilatation, while in unaffected cortical areas their calibre was normal. This was perhaps the most striking feature and being identical with what is found in carbon monoxide poisoning, speaks strongly for stasis as the basis of the tissue damage.

In the second case an apparently total occlusion of a large branch of the middle cerebral artery had caused a fairly sharply demarcated œdema with central necrosis of the cortical grey substance in the regions supplied by branches of the occluded vessels. Microscopically, the general appearance was that of a

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partial œdema of the grey cortex on the basis of a severe ischæmia of the areas involved. By imbibition of fluid into the tissue, the ischæmia had led to a severe damage of the ectodermal elements, but with a slight proliferative reaction. A comparison of these two cases shows how distinct areas of gray cortex can be destroyed by both organic and functional disturbances of the blood-vessels. In the one a demonstrable organic ischæmia of a large pial vessel took place, with rapid and severe necrosis of the supplied tissue. In the other case, a more gradual and less complete disturbance of the nutrition due to stasis resulted.

The authors conclude with the suggestion that the etiology of many obscure transient nervous syndromes and of severe anatomical lesions of the brain without apparent organic cause may be solved by further study of the possibility of functional circulatory disturbances. R. M. S.

[54] **The Boltz (acetic-anhydride) test in cerebrospinal fluid.**—C. THOMAS. *Jour. of Ment. Sci.*, 1930, lxxvi, 271.

OF 23 cases which were Boltz-positive, all were cases of general paralysis and all showed other syphilitic reactions. Of 25 cases which were Boltz-negative, 11 showed positive syphilitic reactions and 14 were completely negative in that respect. Taking the cases of general paralysis for which the Boltz test is supposedly specific, we find that in 34 cases 23 were positive and 11 negative. One is therefore forced to the conclusion that whereas a positive result is an additional confirmation of neurosyphilitic disease, a negative result has no particular significance. Apart from the clinical study of a case with, say, indefinite protein reactions, weak Wassermann or only slightly raised cell-count, the author would be more influenced by the result of the gold-sol test than by either a positive or negative Boltz reaction. C. S. R.

SENSORIMOTOR NEUROLOGY.

[55] **Meningitis associated with occlusion of the spinal cavity** (Méningite accompagnée de d'occlusion de la cavité rachidienne).—DIVRY and LECOMTE. *Jour. de Neurol. et de Psychiat.*, 1930, xxx, 284.

A MAN of 34 fell ill with a meningitis of uncertain nature. The lumbar puncture fluid was characterized by a marked pleocytosis, in which there was at first a polymorphonuclear predominance, but later a lymphocytosis. The patient's temperature fell at the end of a week and the symptoms abated. Weakness of the arms and legs gradually developed, however, about a week later. The tendon jerks became sluggish and the plantar responses were extensor in type. There was retention of urine. Lipiodol was held up between the bodies of the fifth and sixth cervical vertebrae. The cerebrospinal fluid, withdrawn by the

lumbar route, showed a marked increase in protein and a moderate degree of lymphocytosis. Death occurred a few hours after the lipiodol injection, and five weeks after the commencement of the original illness. Autopsy revealed inflammatory changes in the meninges covering the brain, medulla, and upper cervical cord. The dura was adherent to the cord at the site of the hold-up of lipiodol. There was a perivascular type of cerebrospinal meningitis, with a predominantly lymphocytic type of reaction. The white matter of the cord was disintegrated, especially in the region of the posterior columns, where there was a loss of myelin and changes in the axis-cylinders, giving rise to a lacunar appearance. M. C.

[56] **Unusual case of cerebrospinal "giant cocci" meningitis.**—M. H. Weinberg. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 412.

An unusual case of meningococcus 'giant cocci' meningitis is presented from both the clinical and the bacteriological standpoint. Only the finding after a long search of a rare organism and the rapid recovery following serum administration by cistern puncture finally settled the diagnosis. It is desirable to be on the look-out for unusual types of organism in this condition and repeated examinations are indicated. The point of view is stressed that large quantities of serum are not necessary and that successful treatment depends more on ready accessibility of the serum to the disease focus. R. G. G.

[57] **Epileptic manifestations in chronic epidemic encephalitis.**—J. Notkin. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 7.

A discussion of convulsive attacks both cortical and infracortical occurring in epidemic encephalitis. Such attacks are decidedly rare; many of the cases described in the literature either seem to be chance combinations of essential epilepsy and encephalitis or do not belong strictly to the epidemic group.

The occurrence of fits depends more on the extent than on the localization of the encephalitic process. In the infracortical cases the manifestations were very diverse, some being motor, others vegetative, and still others sensori-affective. The various types of these are discussed and illustrated with their bearing on the whole problem of the pathology of epilepsy.

R. G. G.

[58] **Epileptiform convulsions.**—H. L. Parker. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 1032.

In 313 cases of intracranial tumour collected from the Mayo Clinic 67 (21·6 per cent.) were found in which major epileptic seizures had occurred. All of the intracranial tumours associated with convulsions were situated above the

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tentorium. In most of the cases (56) in which convulsions occurred, the tumour was situated in the frontal, parietal or temporal lobes of the brain, in descending order of frequency, or in various combinations of these areas. Convulsions occurred as an initial symptom of the disease in 38 cases. In 13 cases no other complaint had been present for one or more years preceding the development of other signs and symptoms.

In only 29 of the 67 cases did an aura occur as a prodrome to the attack. As might be expected, when present it was either motor or sensory, or both. From the standpoint of diagnosis, the convulsions at some period of the disease differed not at all from those seen in epilepsy, but the tendency toward a change in type is suggestive. A patient may start out with Jacksonian convulsions, and later general attacks may develop, or vice versa. As a factor in the production of convulsions increase in intracranial pressure is fairly well excluded, for infratentorial tumours are more commonly associated with increased intracranial pressure than are supratentorial tumours. The converse also holds, for in the 67 cases in which convulsions occurred, 33 did not disclose ophthalmoscopic evidence of increased intracranial pressure.

R. M. S.

[59] **Occlusion of the aqueduct of Sylvius.**—W. D. Shelden, H. L. Parker and J. W. Kernohan. *Arch. of Neurol. and Psychiat.*, 1930, xxiii, 1183.

From the clinical standpoint, the common feature in a selected group of six cases was the extreme difficulty in reaching any accurate conclusions as to what pathological processes were responsible for the patients' symptoms. It was clearly manifest in all of them that there was an increase in intracranial pressure and probably hydrocephalic distension of the ventricles of the brain. The ages of the patients removed them from the category of patients with congenital hydrocephalus. Most of them had lived apparently normal lives until the first symptoms appeared and definitely suggested a disease acquired after birth. The course was relatively acute in one case, symptoms having been present only nine days before death, but the patients in the other cases had been ill for periods varying from seventeen months to five years. In one case a mistaken diagnosis of suprasellar tumour was made, an error not infrequently due to the symptoms caused by compression of the sella and optic chiasma by the bulging floor of the third ventricle.

In five cases there was a suggestion of hypopituitarism, due, no doubt, to the same mechanical factor. Ataxia was present in three cases; these patients were children aged between 8 and 11 years.

Altogether there was nothing outstanding in the clinical characteristics of the whole group, nor was there anything that would give a reasonable assurance as to the identity of the underlying morbid processes.

From the pathological standpoint, the six cases fall into three groups of two each. In the first group the condition was confined to the region of the aqueduct, was proliferative and resembled in its general characteristics similar changes that have been found going on round the central canal of the spinal cord.

The second group consisted of minute, rapidly growing tumours in a very early stage of formation. They were so situated that before they had reached any appreciable size the aqueduct became inevitably compressed and ultimately occluded. In this group, also, the changes were confined to the region of the aqueduct.

The third group represented a diffuse inflammatory process involving the ependyma of almost the whole ventricular system, with the subependymal tissue. In these two cases it was not definitely ascertained how the infection was acquired, nor was it clear by what pathways such an infection entered the ventricular system.

The authors' cases are interesting, not only for their intrinsic character, but because of the contrast between the almost microscopic size of the lesions and the severe clinical symptoms that finally led to a fatal outcome.

R. M. S.

[60] **Spinal forms of von Recklinghausen's disease** (Les formes spinales de la maladie de Recklinghausen).—L. Cornil and P. Michon. *L'Encéphale*, 1929, xxiv, 765.

The case reported is that of a boy of 15, the subject of cutaneous and subcutaneous neurofibromatosis including a large subcutaneous tumour of the right foot which had been present almost since birth. At the age of 15 symptoms of paraplegia appeared and he was completely paraplegic ' in flexion ' at the time of his admission to hospital. The diagnosis of spinal compression probably by a meningeal neurofibroma appeared to be confirmed by a lumbar injection of lipiodol which was held up at the level of the tenth thoracic vertebra. Following on this injection power and sensation gradually returned to the legs, until he was able to walk for two or three kilometres. Thereafter some increase of stiffness set in, but he remained able to walk. In view of this improvement the diagnosis of spinal tumour seemed less certain, and the authors considered the possibility that the paraplegia might have been due to bony deformity in the spine, but found no evidence of this. Shrinkage of a spinal neurofibroma under the prolonged action of lipiodol appeared to them a possibility in view of the œdematous nature of many of these tumours. The later progression of symptoms certainly seems to be in favour of this hypothesis.

J. G. G.

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[61] **The incidence of fever and leucocytosis in multiple sclerosis.**—J. B. McKenna. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 542.

The author investigated the occurrence and incidence of abnormalities of body temperature and of leucocyte count in 109 cases of disseminated sclerosis.

In 40.4 per cent. there was neither fever nor leucocytosis, but three showed increased cell counts in the cerebrospinal fluid; 55.9 per cent. showed elevations in temperature, 44.9 per cent. between 99° and 99.5° F. and 11 per cent. between 99.6° and 100° F.; 22.9 per cent. showed definite leucocytosis, 3.6 per cent. occurring without elevation in temperature and 19.3 per cent. accompanying febrile reactions. Pleocytosis of the cerebrospinal fluid occurred in nineteen cases, or 17.4 per cent.; three of these cases showed normal temperatures and leucocyte counts; seven were accompanied by fever alone; two by leucocytosis alone, and seven by both fever and leucocytosis.

R. M. S.

[62] **Myatonia congenita**—E. S. Gurdjian. *Arch. of Neurol. and Psychiat.*, 1930, xxiv, 52.

Several cases of myatonia congenita are reported. In one case in which an autopsy was performed microscopic examination of the nervous system and striated muscle indicated involvement of both. There was marked change in the anterior horns and abnormalities in the muscles which could hardly be merely the reaction to pathological changes in the anterior horns. The author suggests that a common factor, and probably a toxic one, involving the neuromuscular system may be responsible. In favour of this assumption are the postnatal cases of this disease with an onset usually after an acute infection.

R. M. S.

PROGNOSIS AND TREATMENT.

[63] **Epilepsy: clinical observations on the control of convulsive seizures by means of dehydration.**—T. Fay. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 481.

A very long article reviewing recent work on epilepsy and advancing the theory that fluid control and water balance play a large part in the cycle of convulsive attacks and that the major forms of the seizures can be modified or controlled with proper regulation of these factors. The author thinks that epilepsy (as well as other mental deficiencies) has an understandable hydraulic pathology and that certain structural traumatic and inflammatory considerations are present in the majority of these conditions. Full consideration must be given to the physiological and vasomotor factors which complicate the problem, and an exact means of study by encephalography and all other methods at our

disposal must be applied to the solution of every case. It is evident that there cannot be a ' cure ' of what appears to be a normal physiological response under certain conditions ; the control of the factors surrounding the production of this response must include all of the accepted methods—whether medical, surgical, dietary or dehydrating depends on the indications furnished by the patient's case. We may thus expect amelioration of the seizures or their control so long as the individual's ability to maintain and compensate a normal life in the presence of deficient structural and physiological mechanisms makes it possible to do so.

The author is convinced that the predisposing factor concerned with the major but not necessarily with the minor attack is due to a hydration state however induced—whether local or diffuse—and that proper control of this factor whenever possible gives rise to an attack-free existence, which may continue so long as the patient's water metabolism or fluid balances remain within the limits of proper compensation.

R. G. G.

Psychopathology.

PSYCHOLOGY.

[64] **Observations on the temporal judgment during sleep.**—Edward N Brush. *Amer. Jour. Psychol.*, 1930, xlii, 408.

The ability to awaken at given times in the morning is one approach to the problem of the temporal judgment during sleep. The writer here records the results of his experiments in this direction. His procedure was to record, on retiring, the approximate time of going to sleep, the time chosen for awakening, and ratings on such factors as general physical condition, degree of mental alertness previous to retiring, motivation, and the amount of sleep on the previous night. Then the suggestion for awakening was given by repeating ten times subvocally : ' Waken me up at x o'clock.' The time of awakening was taken as the time when consciousness was sufficiently regained to look at the clock, and it was recorded to the nearest minute. Other conditions recorded were : illumination in the room, dreams, general physical condition during the first half-hour after awakening, and the ' purity ' of the determination (i.e. the amount of subsequent mental activity before dropping off to sleep). It was found possible to ' judge ' time with considerable accuracy during a period of sleep. The ' judgment ' appeared as awakening under a determination. The average actual time of awakening was far closer to the time set in the experiment than to any time, absolute or relative, that one might have expected for the awakening on the basis of habit. Certain conditions appear as favourable

or unfavourable to the operation of the determination. The more important of these were general physical condition, amount and character of sleep, mental activity subsequent to the setting up of the determination, both before and after going to sleep, illumination in the room on awakening, and motivation.

C. S. R.

[65] **The psychology of imitation with special reference to early childhood** — C. W. VALENTINE. *Brit. Jour. Psychol.*, 1930, xxi, 105.

THE wide divergence of the views of leading psychologists about imitation demands a thorough enquiry into the facts as to the beginnings of imitation during infancy. From his own observations Valentine concludes that actions to which there is already an innate tendency are imitated very readily within the first few months, e.g. sound-making and smiling. Actions which can serve no purpose perceived by the child and which are not based on instinctive impulses, are imitated freely between the ages of nine and twelve months. There is sometimes a 'latent' period between the seeing of an action and the imitation of it, and sometimes the repetition of an action is necessary before imitation follows. Primary, involuntary, or purposeless imitation seems to be due to the monopolization of attention for a moment by some fascinating impression. Some imitations seem to serve the purpose of helping the subject to realise or enter into the experience of the imitatee more vividly. Some imitations are of a reflex type, if the term reflex can be applied where sight provides the only stimulus. Experimental tests at twelve months and at two years suggest that the tendency to imitate any interesting action is strong in some children. The imitatee is important: a child will sometimes imitate its mother but not another person. There is some evidence that a child may wish others to join in the imitation. The evidence as a whole seems conclusive against the view of some *Gestalt* psychologists that imitation is always purposive. C. S. R.

[66] **The biological significance of blushing and shame.**—JOHN T. MACCURDY. *Brit. Jour. Psychol.*, 1930, xxi, 174.

A NUMBER of authors have concluded that the behaviour accompanying the emotion of shame represents an instinctive seeking of cover. Blushing is, perhaps, an evidence of the shifting of balance in the involuntary nervous system towards a strengthening of the vagal component at the expense of the sympathetic. This suggests a reaction by the organism towards inactivity and, among the danger responses, this would mean immobility rather than flight or aggression. Immobility among animals turns to hiding in man, and the physiology of blushing and the behaviour of shame are thus identified at primitive levels. Among savages concealment is sought for practices that are

potentially dangerous in a hostile environment. These are eating, sleeping, sexual intercourse and excretion. Shame develops in connection with publicity in the indulgence of these four. In modern civilization security has been achieved for the first two and only traces of shame persist in connection with them. Modesty, of biological origin, has over-determined the shame concerning sex and this has been extended to excretion. Finally, symbolism has made possible the appearance of shame in connection with practices that are neither sexual nor potentially dangerous. C. S. R.

[67] **Jealousy.**—Ernest Jones. *Psyche*, 1930, xi, 41.

Jealousy is a much less normal phenomenon than is commonly supposed. It is the writer's experience that for the greater part it rests on an abnormal and neurotic basis. It betokens a failure in the development of the capacity to love, a lack of self-confidence due ultimately to unconscious guiltiness that has not been overcome from childhood days, and an undue dependence on the love object that indicates a tendency in the direction of sex inversion. This last feature becomes plain enough in insane jealousy but it is present in a milder degree in the other forms also. In short, jealousy is a sign of weakness in love, not of strength ; it takes its source in fear, guilt and hate rather than in love. C. S. R.

[68] **The relation of extroversion-introversion to intelligence and tuberculosis.** —Clarence A. Neymann. *Amer. Jour. Psychiat.*, 1930, ix, 687.

Nine hundred normal individuals were examined by the Neymann-Kohlstedt test. Most individuals fell into one of the two groups, the introverts and the extroverts. It was concluded that intelligence and introversion do not coincide and that the average tuberculosis patient has a strong leaning towards introvertive qualities. The introvertive qualities were found to decrease as pulmonary tuberculosis became progressive and the patient bed-ridden.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[69] **Neurogenic and psychogenic disorders.**—J. L. Kantor. *Jour. Nerv. Ment. Dis.*, 1929, lxx, 42.

A survey of the literature on the subject seems to show that an increasing number of cases formerly diagnosed as nervous dyspepsia are demonstrated on study by improved methods to be instances of actual organic disease of the digestive organs or of their nervous communications. The autopsy findings

of post-inflammatory nerve lesions in cases of digestive disorders suggest the advisability of careful scrutiny of dyspeptic patients for evidence of past infections (such as tuberculosis) which might involve important nervous pathways in scars or adhesions. The theory of autonomic imbalance occupies a prominent place in discussions of the etiology of such digestive disorders as peptic ulcer, cardiospasm, intussusception, and congenital pyloric stenosis.

In some digestive conditions (intestinal tuberculosis, some cases of peritonitis) it is the secondary nerve changes and not the primary lesion which produce the characteristic symptomatology.

The concept of solar syndromes, with or without actual lesions in the nerve plexuses, is advanced by some writers to explain the painful phenomena in a rather diverse group of abdominal conditions.

Lesions of the spinal cord may produce visceral effects either directly by involving the preganglionic fibres at their point of origin or else indirectly by affecting the suprasegmental neurones connecting with these preganglionic fibres. The general result of destroying the superior connections is a release of the lower neurone reflexes.

Neural ileus is an important condition often leading to useless surgical exploration. This form of ileus may be due to lesions either in the segmental or suprasegmental arcs or to psychoses with marked depression. It is a common symptom of transverse myelitis.

Important centres regulating the functions of the entire involuntary nervous system are now known to be located in the diencephalon. Lesions in this region occur frequently in epidemic encephalitis, dementia paralytica and dementia precox. Psychical disorders of digestion are mediated through the emotions and through ideas causing conditioned reflexes. Conditioned reflexes, therefore, furnish a basis for the development of digestive habits, peculiarities, and perversions. The psychoneuroses are not uncommonly encountered by gastro-enterologists because the digestive symptoms are prominent and varied. In melancholia, constipation is a particularly constant and stubborn manifestation. R. G. G.

PSYCHOSES.

[70] **A brief résumé of the types of insanity commonly met with in India, with a full description of the "Indian Hemp insanity" peculiar to the country.**—Jal Edulji Dhunjibhoy. *Jour. of Ment. Sci.*, 1930, lxxvi, 254.

According to the last census, the proportion of insane to general public in India is 28 or 29 per 100,000, but there is no reliable information as to the number of uncertified cases. Manic-depressive psychosis contributes largely

to the population of mental hospitals. Dementia præcox takes the next place in number and the more highly educated seem to be most prone to this form of psychosis. The toxic psychoses are next in order of frequency. In this group hemp drug heads the list and alcohol is second. Acute confusional insanity is also not uncommon in India and in a good many cases the principal etiological factors are starvation and physical exhaustion. The psychoses associated with infections, pregnancy and the puerperium are also common. Mental defectives take the fourth place. General paralysis is extremely rare in India though syphilis is very common. The writer has not yet seen such a case in an Indian who has not been abroad. Various theories have been propounded for this infrequency of general paralysis in India, but none so far is convincing. Indian hemp intoxication varies in degree. Some become drowsy or semicomatose, rapidly passing into a dreamy state, with a rapid flow of ideas, often of a sexual nature, ending in a deep sleep. Others go through a phase of increased psychomotor activity before passing into the dreamy state. Large doses produce excitement, delusions, hallucinations, rapid flow of ideas, a high state of ecstasy, increased psychomotor activity, with a tendency to wilful damage and violence, to be followed by deep sleep and forgetfulness of all but the initial symptoms. Acute mania, chronic mania, and dementia, are the three types of hemp insanity most commonly met with. A peculiar eye condition is almost pathognomonic of it; there is a marked conjunctival congestion seen in the horizontal vessels of both eyes. The prognosis is hopeful. In acute cases nearly 90 per cent. and in chronic cases 40 per cent. recover. Stoppage of the drug produces no abstinence symptoms. Excessive or prolonged use of hemp drugs degrades the mind and character of the consumer and predisposes him to commit crime.

C. S. R.

[71] **Mental disorders from the use of hashish** (Les troubles psychiques dus à l'emploi du haschisch)—Fahreddin Kerim. *L'Hygiène mentale*, 1930, xxv, 93.

As in alcohol, the influence of hashish varies in different personalities. Its principal effect is an enfeeblement of the organism. There is produced a general lassitude; movement is slow; will-power is much limited; and the association of ideas lessened or severely disturbed. The patient soon ceases to interest himself in his human environment, his imagination becomes exalted and completely self-absorbed, and he revels in all sorts of dreams. Later, ideas of time and place are lost, speech is stammering, movements are made without any confidence, and helplessness is prominent. Others present a different picture, becoming delirious with impulsive violence and hallucinations. Crimes may be committed while in this state. Such a condition may be observed in those who have commenced to take the drug in doses of 20 to 30 centigrammes. In others

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these manifestations do not appear until the dose taken is 3 grammes. Certain hashishists with a minimum of intoxication by the drug imagine that they have reached the acme of their desires, believe that they are great and rich and tell of the joys they have experienced in their hashish dreams. There are those, too, who on the contrary fall into a profound apathy. They are possessed by fear and ideas of persecution torment them. Physically, the expression is dull, there is a general anæmia and perhaps a yellowish complexion. There is an exaggeration of the tendon reflexes, dyspnœa, and increased cardiac movements. The main mental disorders which are met with in inveterate hashish takers are cerebral excitement, subacute melancholia, dementia præcox, a schizoid state, an auditory hallucinated delirium, and mental confusion.

C. S. R.

[72] **Manic-depressive psychosis in identical twins.**—Isabel A. Bradley. *Amer. Jour. Psychiat.*, 1930, ix, 1061.

A case is here recorded and it is noted that the significance of such striking similarities in structure as these twins presented associated with equally striking similarities in symptoms of a psychosis is of interest and importance. It is very strong evidence that the cause of this type of psychosis and probably of all psychoses is an inherent defect in the germ-plasma, a germinal inheritance that makes the development of a psychosis possible. There is no case on record so far as the writer knows of dementia præcox in one only of identical twins. Mongolism, it is stated, has not been observed in one of identical twins. Epilepsy has not been reported in one identical twin.

C. S. R.

[73] **The relative importance of hereditary factors in manic-depressive psychosis and involutional melancholia.**—C. B. Farr, P. Sloan and L. H. Smith. *Jour. Nerv. Ment. Dis.*, 1930, lxxi, 409.

The authors tabulate their findings in accordance with a schedule of familial psychopathic factors and find that manic-depressive cases show considerably more family taint than the involution cases.

R. G. G.

PSYCHOPATHOLOGY.

[74] **The incidence of congenital syphilis in an institution for the feeble-minded.**—Chas. S. Woodall. *Amer. Jour. Psychiat.*, 1930, ix, 1065.

The Wassermann, Kahn, and Hinton reactions were applied to 1,314 feeble-minded patients and many retests were done. The Hinton was found to be

twice as sensitive as the other two tests. The diagnosis of syphilis was based upon serological and clinical findings and the percentage obtained was 7·2 per cent. Congenital syphilis was present in 6·5 per cent. of the cases. Clinical as well as serological evidence is of great value. Defective heredity was present in 16 per cent. of the congenital luetics and this factor lowers the figures of incidence. The writer believes that certain cases of mental defect are due to congenital syphilis. How many are thus caused has not been established, nor has the nature of the disease-process been elucidated.

C. S. R.

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Reviews and Notices of Books.

Psychoanalysis of the Total Personality. By FRANZ ALEXANDER. New York: Nervous and Mental Disease Publishing Co., 1930. Pp. 176. Price $2.50.

IN this book Dr. Alexander endeavours to apply in a practical manner to the neuroses those theories of Freud which are set out in the Ego and the Id. This he does in an admirable way and his presentation is so clear that even those who are not initiated into the Freudian vocabulary will be able to follow it with interest and profit. Those who approach the study of the neuroses from a neurological standpoint will no doubt find much to criticise, notably the assumption of the capacity of the infant to undertake mental tasks of considerable intricacy which are presumably conscious at this stage and are only repressed into unconsciousness during the beginning of the latency period. It would be of great interest and importance to discover whether the child of under five really has enough fully developed neurones at his disposal for this purpose; the recovery of these 'memories' under analysis is not altogether convincing evidence. The other point is the extreme personalization of the divisions of the personality, which however, Alexander fully admits and deliberately adopts for purposes of description.

The personality is separated into three divisions under this scheme: (1) the conscious ego, (2) the purely instinctive id, and (3) the still unconscious superego, which represses the socially undesirable demands of the id. In large measure conscious and environmental repressions are handed over to this unconscious superego much as walking is handed over to lower centres once the process is learnt.

In the normal subject there is a reasonable balance between the demands of the id and the claims of the superego, so that the former are sublimated into desirable activities. In the neurotic there is over-repression by the superego, so that there is always a demand of the instinctive strivings (the id) for satisfaction and a demand of the superego for punishment. The symptoms of the neurotic therefore always have a double purpose, an instinctive wish-ulfilment and a self-punishment. In conversion-hysteria this is fulfilled at one and the same time, the symptoms being at once an indirect gratification of the id and a punishment of the self by restricting the activities of the individual. In compulsion neuroses there is a highly unstable equilibrium between the indirect satisfaction of the id and the punishment demands of the superego. In manic-depressive psychosis there is an alternating preponderance of id (manic stage) and superego (depressive stage), which accounts for the tendency

for suicide in the latter stage ; the self-punishment corresponds to the death-instinct (Thanatos) and the id to the life-instinct (Eros) of Freud. There is an interesting discussion of the extroverting socializing influence of 'Eros' and the introverting individualizing influence of 'Thanatos.' Altogether this is a brief, clear and commendable exposition, and all serious students of the neuroses ought to study it with care. R. G. G.

Reflex Action ; a Study in the History of Physiological Psychology. By FRANKLIN FEARING, Ph.D., Northwestern University. London ; Baillière, Tindall and Cox. 1930. Pp. 350. Price 30s.

PROFESSOR FEARING has laid the neurologist and psychologist under a debt of gratitude for his minute historical survey of the vicissitudes undergone by the conception of reflex action. Any close reader of the scientific literature of the day in these two subjects must be impressed by the looseness of thought characterising use of the term in many instances, and by its extension to cover all kinds of movement. With a view to providing a thoroughly reasoned basis the author takes us seriatim from Descartes and Vesalius right down to the present, to the day of Pavlov and Sherrington. He cannot give a simple and unqualified definition, but allows a threefold classification. (1) Reflex action is involuntary, unlearned, predictable, invariable, purposive, and does not involve the cerebral cortex neurally. This is the 'specific' concept. (2) The 'genetic' concept declares it is derived from voluntary action through the intermediary of impulsive and automatic action. (3) According to the 'mechanical' concept *all* behaviour is predetermined by reflex neural arrangements. 'Choice' and 'purpose' disappear as factors. Terminological difficulties are ever in evidence in connexion with this analysis, and, be it once more stressed, artificialities. Division of arts into volitional, automatic, habitual, reflex, etc., makes an 'artificial stratification of animal responses.'

Nouveau Traité de Psychologie. Edited by GEORGES DUMAS, Professor at the Sorbonne. Paris : Felix Alcan. 1930. Pp. 430. Price 75 fr.

THE first volume of a new system of psychology, to be completed in ten, has made its appearance. Edited by Professor Dumas of the Sorbonne, with the assistance of a large number of distinguished collaborators, it promises well if we are to judge by this first instalment, which is concerned mainly with prolegomena. Successive chapters deal with the place of man in the animal series, with anthropology, with the physiology of age and sex, the general and special aspects of neurophysiology, consciousness from a biological viewpoint. After these introductory matters the subject proper begins with a discussion of the objects and methods of psychology.

There is much of interest to the philosophical neurologist and to the psychiatrist in these pages ; the summaries of present-day knowledge as regards the functions of the nervous system and the relation of

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' psychism ' to various neural levels are clear and informative, while the chapter on consciousness is particularly illuminating. The general tenor of the more specific psychological chapters in this first volume finds expression in the initial sentence of the editor's introduction : " Une conception commune à tous les collaborateurs de ce Traité . . . consiste à considerer la psychologie comme uniquement fondée sur des faits et à exclure, par la même, de son domaine, toutes les speculations ontologiques."

Biological Principles. By J. H. WOODGER, B.Sc., Reader in Biology in the University of London. London : Kegan Paul & Co. 1930. Pp. 498. Price 21s.

THE main thesis of this ' provocative ' if not provoking book, caustically critical of much that passes for biological doctrine to-day, is that recent progress and acquisitions in the realm of physics (more particularly) should be applied to biological thought. Explanations that do not explain, antitheses that mutually exclude, teleological notions utilised in biology, rival theories of development, all come under the survey of the author, who does not spare the rod when considered salutary, and who certainly possesses a penchant for seeking out the flaws in the armour of the combatants in the field. His book is as a fact highly informative, mentally stimulating, and particularly useful in its succinct exposés of current hypotheses and views. What is largely novel and personal are his efforts to apply to his own subject various present-day discoveries from other domains of thought and research. For example, he tilts at the anatomist for studying (in what is usually believed to be a concrete science) what are really a series of abstracta. The ' heart ' of the anatomist is a ' spatial ' heart, ' much more abstract than the heart as it is in the living body.' The ' visual ' heart in reality is itself an abstractum, ' a synthesis of many visual appearances of the heart as it appears after pickling.' What the author endeavours to convey to his reader is, that ' the heart is an event.' To those who can accept the contention in the statement that ' anatomy is and must be very abstract ' this line of thought will doubtless appear enlightening, and they may perhaps be willing to follow the writer in his further argument, that the concrete organism is a spatio-temporal structure, and that this structure *is* the activity of the organism. With other words, in nature no antithesis between structure and function exists.

Inversions-atmung und Geisteskrankheit. By Dr. med. OTTO BUSINGER. Munich : Ernst Reinhardt. 1930. Pp. 48. Price Mk. 3.50.

ANY light that can be thrown on the secret of dementia præcox must be welcome. Dr. Businger approaches his subject by sketching in a few paragraphs the fundamental importance of respiration in the bodily economy and metabolism. Parallelism of the respiratory current is a physiological necessity for proper aeration and oxygenation. He then states that he has examined

some 500 cases of mental disease (mainly dementia præcox), and among them has found in a high percentage definite respiratory insufficiency, due usually to defective air-entry. He says that the average respiration rate per minute of cases of that psychosis is 18—21, whereas that of normal subjects is 16—19. To this impairment, and all that it must entail by way of retarded and inadequate metabolism, Dr. Businger ascribes many of the symptoms of the affection, though at this point we think his interpretations occasionally become a little strained, as when he asserts that the catatonia of the dement is adopted to counteract the effects of respiratory fault. On many cases he has operated, dealing with the nasal passages so as to procure better aeration, and he claims immediate improvement of a semiological kind.

There may be something in all this, possibly much. At any rate the slender monograph is evidently based on personal experience, is well documented, and written without any trace of overstatement. It merits consideration.

Pleasure and Instinct. By A. H. B. ALLEN. London: Kegan Paul & Co. 1930. Pp. 336. Price 12s. 6d.

IN an ingenious and readable fashion the author examines what is known of the affective side of mental life. The 'feelings' are regarded by some as akin to sensations, by others as a different order of mental facts, arising in the course of conative activity. Here the view is adopted that pleasure and pain, or unpleasure, are dependent on the success or otherwise of conation; the whole mental life of man is held to consist of a 'striving process,' included in which is adequate function of the instincts. With their activity feelings are coupled. According to the author the chief conscious instincts can be shewn to be derivatives of the primitive urge to 'self-maintenance.'

From the standpoint of pure psychology the argument is cleverly elaborated; nevertheless there is a considerable disadvantage in his not endeavouring to correlate neurological and, for that matter, psychopathological disorders affecting the sphere of the feelings with what is deduced largely by introspection. Little cognisance is taken of the data supplied by these studies favouring the view that feeling is connected with the activity of certain definite cerebral mechanisms.

The Meaning of Sacrifice. By R. MONEY-KYRLE, M.A., Ph.D. The International Psycho-analytical Library (No. 16), edited by ERNEST JONES. London: Hogarth Press. 1930. Pp. 273. Price 18s.

A NOTEWORTHY addition to psycho-analytical literature is formed by Dr. Money-Kyrle's thesis on sacrifice and its meaning, which is conceived along historical lines in general, and the subject examined in the light of accepted psycho-analytical doctrine. The relation of sacrifice to parricide, expounded to some extent by Freud, is not considered by the author of necessity to entail

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acceptance of the theory of a 'racial mind' or of the 'inheritance of acquired memory'; and he develops with logical minuteness his own view, that it is the 'symbolic expression of an unconscious desire for parricide which each individual has himself acquired.' The Œdipus complex is capable of a number of variations, in accordance with different impulses at different levels of the mind. Thoughtful in conception and clear in exposition, the book deserves careful study.

Die Röntgenbehandlung der Nervenkrankheiten. By Prof. Dr. Otto Marburg and Doz. Dr. Max Sgalitzer, Vienna. Berlin and Vienna: Urban and Schwarzenberg. 1930. Pp. 213. Price not stated.

To the average English neurologist it will come rather as a surprise, we believe, to learn the extent to which the application of X-ray treatment in nervous disease has been advanced in the Viennese school, and elsewhere too, for that matter. If we are to judge by the data herein provided, there would appear to be practically no variety of nervous affection, peripheral or central, acute or chronic, in which radiation has not been given a trial. Naturally, its use has been recognised mainly in connexion with neoplasms, whereas the authors furnish details of its employment in conditions as widely separated as tabes and epilepsy, hydrocephalus and torticollis. If its adoption for some conditions has been purely empirical, for others perhaps more rational, in all it has been experimental; and the value therefore of the record is considerable even in its negative aspect.

Recent Advances in Neurology. By W. Russell Brain, M.D. (Oxon.), M.R.C.P. London, and E. B. Strauss, B.M., B.Ch. (Oxon.), M.R.C.P. London. Second edition. With 39 illustrations. London: J. & A. Churchill. 1930. Pp. 429. Price 12s. 6d.

That a second edition of this book should be required within a comparatively short period is indicative of its practical usefulness and relative completeness. Such changes as the new edition carries refer chiefly to blood-vessel tumours of the brain, varieties of encephalitis, the serum treatment of acute poliomyelitis and the malarial treatment of general paralysis. Ketogenic diet-sheets are also included. Clearly written and well considered, the material herein embodied is exactly that about which the keen practitioner will seek information, and to him the volume is once more recommended.

Psychiatrie du Médecin Practicien. By M. Dide and P. Guiraud. Second edition. With 8 plates. Paris: Masson et Cie. 1930. Pp. 465. Price 45 fr.

Written for the student and practitioner, in clear, concise, and graphic language, this little handbook of psychiatry is excellently done. It forms a collection of symptoms and syndromes as encountered at the bedside, and

says little of classification, of nosology, of disease-entities. Etiology and pathology, however, receive a share of attention, and treatment too. The semiological arrangement of the book is obviously based on firsthand clinical observation of an attractive kind, while the illustrations in collotype are unusually adequate ; there is also (rare in French publications) an alphabetical index to facilitate reference. With this at hand, as an introduction to more ambitious textbooks, the student of psychiatry is well provided for.

Manuel Élémentaire de Psychiatrie. By M. NATHAN. Paris ; Masson et Cie. 1930. Pp. 319. Price 30 fr.

FOR his first contact with mental disease this slender volume is offered the medical student. It embodies present-day views concerning the psychoses and, unhampered by controversial questions and terminological discussions, is content to furnish him with a precis of clinical psychiatry. Within its obvious limits it seems sufficient, but some of the illustrative figures (e.g. fig. 27) cannot be considered serviceable.

The Influence of Habit on the Faculty of Thinking. By MAINE DE BIRAN. Translated by M. D. BOEHM. With an introduction by GEORGE BOAS, London ; Baillière, Tindall and Cox. 1930. Pp. 227. Price 22s. 6d.

THIS is the third volume to appear in the series of reprints and translations forming the ' Psychology Classics ' edited by Professor Knight Dunlap. In French psychology the work of Maine de Biran, published more than a century ago, has always been held in high esteem for his pioneer study of habit as a psychological rather than a physiological function, and also as the starting-point of that French spiritualism which culminated in the philosophy of Bergson. For these among other reasons this translation of his work will prove of interest to the student of psychological movements.

Male Disorders of Sex. By K. M. WALKER. London : Jonathan Cape. 1930. Pp. 191. Price 5s.

IN this little book the author has reviewed in readable form the various problems about which the doctor is asked by his male patients. Impotence, homosexuality, masturbation, emissions, sterility and other subjects are dealt with shortly but informatively. A just balance is kept between physical and psychical factors both in etiology and treatment. The psychoanalysts will no doubt be dissatisfied that he does not consider their method the only treatment for psychical impotence, and in many respects the enquirer will desire fuller information than can be given within the compass of such a small volume. As an introduction to the subject, however, this book can be thoroughly recommended.

R. G. G.

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The Morphine Habit and its Painless Treatment. By G. LAUGHTON SCOTT, M.R.C.S., B.A.(Oxon.). London; H. K. Lewis and Co. 1930. Pp. 95. Price 5s.

DR. LAUGHTON SCOTT is an enthusiastic advocate of the method of gradual reduction in cases of narcotic addiction, and has published an account of his particular technique which for clarity and practicability leaves nothing to be desired. Briefly, it consists (in a limited sense) of a modification of the Lambert method; tolerance to belladonna and hyoscine is gradually developed, with free and increasing use of luminal. In the author's hands the results have been very satisfactory, time consumed in treatment relatively short, discomfort and distress minimal or absent altogether.

La Folie et la Guerre de 1914-1918. By A. RODIET and A. FRIBOURG-BLANC. With ten plates. Paris: Felix Alcan. 1930. Pp. 194. Price not stated.

INTERESTING questions of cause and effect are raised by this graphic account of the psychopathology of the War, which is illustrated by a fine little series of clinical photographs. With many another, the two experienced psychiatrists who write it agree that the War originated no new psychopathological syndromes, and that its major influence was exerted in the direction of accentuating known etiological factors and modifying symptomatological evolution.

Monographs on Experimental Biology. Edited by JACQUES LOEB, Rockefellar Institute; T. H. MORGAN, Columbia University; and W. J. V. OSTERHOUT, Harvard University. London: J. B. Lippincott Co. V.Y.

WE have received for review the following volumes in the above-mentioned series, viz. *Labyrinth and Equilibrium*, by Dr. S. S. Maxwell; *The Elementary Nervous System*, by Dr. G. H. Parker; *Smell, Taste, and Allied Senses in the Vertebrates*, by the same author; and *Physical Basis of Heredity*, by Dr. T. H. Morgan. Priced at 12s. 6d., these volumes, which were first published some time ago, are of especial interest to the student of neurology who appreciates the importance of some knowledge of the comparative science as a means of aiding interpretation of nervous semiology in hiuman subjects. Biologically old mechanisms in the latter are often laid bare by disease, and their activities can be better realised by the light of information as to their performance in animals lower in the scale. For this among other reasons the series should be in the hands of all neurologists who seek to learn more of the physiology of the nervous system.